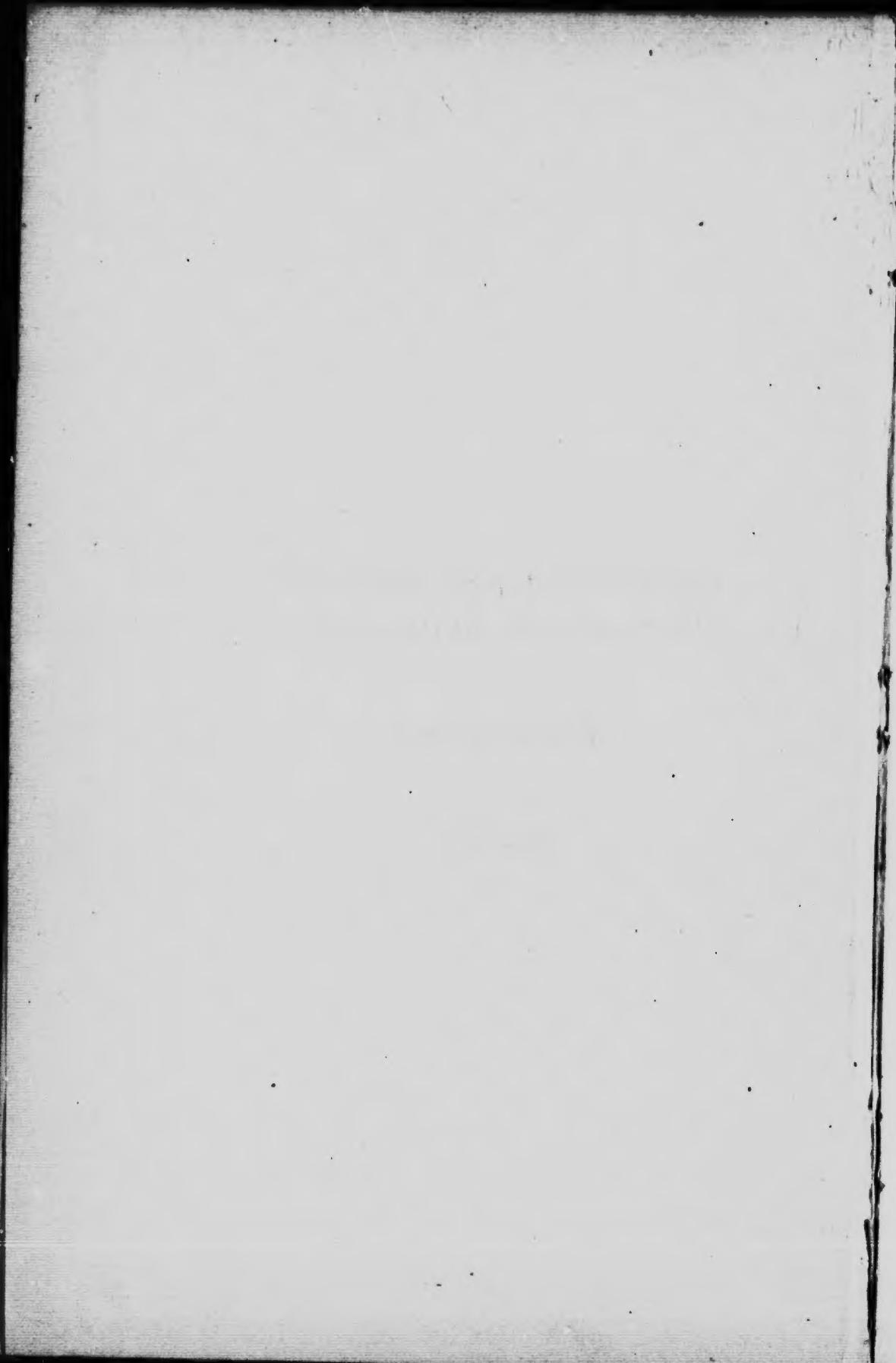


**FRIEDBERGER AND FRÖHNER'S  
VETERINARY PATHOLOGY**

**[Authorised Translation]**

**VOLUME II**



# Friedberger & Fröhner's Veterinary Pathology

[Authorised Translation]

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VOLUME II

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## P R E F A C E .

THERE has long been wanting in Veterinary Literature a comprehensive text book dealing with the Diseases of Animals from their clinical, pathological and therapeutical standpoints, and which might have some claim to the term "modern." This gap was efficiently filled in Germany by Friedberger and Fröhner's splendid production, the value of which was soon correctly estimated by Veterinary Surgeons of all nations. The late Captain Hayes, the most prolific English Veterinary writer of modern times, and editor of many valuable works on veterinary and equine matters, ever quick to appreciate merit, bought the right to translate the work into English. He chose to divide the subject into two volumes, the first of which, "Infective Diseases of Animals," he lived to see run through two large editions. Although not quite sure that this was quite the best division which might have been made, I feel bound to call this second volume "Non-infective Diseases of Animals." Captain Hayes, at the time of his death, was at work on this volume, and I was then requested by Mrs. Hayes to undertake the completion of the task. Being at the time a teacher in a Veterinary School, I greatly felt the need of the book, and being anxious to see it completed, I consented. With the able assistance of Mr. G. A. Page and Mrs. Hayes, I have endeavoured to make this volume as accurate as possible, making slight additions and corrections in some few places where recent discoveries have made it necessary. As the book is intended for English-speaking Veterinary Surgeons, I have deemed it advisable to give all weights and measures in English.

JOHN DUNSTAN.

I, Dean Terrace,  
Liskeard, Cornwall.  
May 1st, 1905.



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## CHAPTER I.

### DISEASES OF THE ORGANS OF DIGESTION.

#### STOMATITIS (INFLAMMATION OF THE BUCCAL MEMBRANE).

**Forms.**—Inflammation of the mucous membrane of the oral cavity appears in various forms. In the first place we have a primary, independent and idiopathic stomatitis in contradistinction to symptomatic or secondary stomatitis, which is a manifestation of certain general diseases, such as foot and mouth disease. Also, according to the respective causes, we recognise a traumatic form, which is produced by mechanical, chemical, or thermal irritants; and an infectious, mycotic, gregarious and toxic form, which includes the pustular and contagious stomatitis of horses, the thrush of birds, the croupy-diphtheritic stomatitis of birds, and mercurial stomatitis of cattle. Finally, according to the anatomical changes of the mucous membrane, we may divide stomatitis into erythematous, catarrhal, erysipelatous, phlegmonous, aphthous or vesicular, pustulate, ulcerous, croupy and diphtheritic forms.

From a pathological point of view, it is best to distinguish the independent clinical varieties of stomatitis as follows :

1. Catarrhal.
2. Aphthous or vesicular.
3. Ulcerous.

#### I. STOMATITIS CATARRHALIS (CATARRHAL INFLAMMATION OF THE BUCCAL MEMBRANE).

**Etiology.**—Catarrhal stomatitis is the mildest form of inflammation of the mucous membrane (simple catarrh), and is caused by the action of various irritants. Among traumatic irritants we have very rough and hard fodder, chaff, sharp

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bits, injurious manipulations within the oral cavity, and foreign bodies. Among chemical irritants in the case of herbivora, there are a great number of poisonous plants, such as aconite, hellebore, ranunculus, euphorbia, daphne, colchicum, corn-poppies, tobacco, corn-cockle, digitalis, yew, spotted hemlock, water-hemlock, fool's parsley, rhododendron, charlock, etc. Catarrhal stomatitis may be produced by the inadequate dilution of certain therapeutic agents, such as electuaries containing tartar emetic; solutions of corrosive sublimate; ointments containing arsenic, chloride of zinc and other substances which can be licked off; and solutions of caustics and acids. Pungent spices (pepper for instance), and the skins and roots of black radishes (*raphanus sativus*) produce a similar effect. Thermal irritation is caused by the consumption of hot food and drink.

The presence of pathogenic vegetable germs in fodder is another cause of catarrhal stomatitis. These fungi frequently produce not only a catarrhal, but also a vesicular and ulcerous inflammation. The "rape-destroyer" (*polydismus exitiosus*) often produces severe stomatitis in cattle and sheep. *Puccinia graminis* (common grass and corn blight), *puccinia arundinacea*, and the uromyces fungus, which lives on Swedish clover, have a similar action. Among the rusts, bunt (*tilletia caries*), which was formerly called *uredo foetida*, or stinking rust, plays a prominent part. Mould-fungi (as in mouldy oats) and mildew (*erysibe communis*) are not potent producers of this disease. These fungi have essentially a chemical action (toxine).

The consumption, along with food, of the hairs of caterpillars is a special cause of stomatitis. The hairs of the *bombyx s. cเนethocampa processionalis*, and of the hawthorn caterpillar (*porthesia chrysorrhæa*). Besides the mechanical irritation, it is probable that a chemical poison (enzyme or formic acid) is also in action. For instance, Kösters saw 52 artillery horses ill on account of their green food having become tainted by hawthorn caterpillars. In a similar way, plant lice (*aphis*) produce stomatitis in herbivora.

Apart from causes which have a directly irritating effect on the oral mucous membrane, stomatitis occurs in all the domestic animals, and especially in horses, from unduly prolonged abstinence from food. This form of the disease is particularly well marked in digestive disturbances, in which cases the cast-off epithelial masses, which are usually removed during the consumption of food, become decomposed, and consequently set up

## STOMATITIS CATARRHALIS.

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inflammation of the buccal membrane. The same remark applies to chronic general diseases of a febrile type, in which there is the further aggravating circumstance that the digestive secretions (saliva in this case) become decreased in quantity and morbidly changed, which fact increases the liability of the desquamated epithelium to become decomposed. A special form of stomatitis (stomatitis mercurialis) is found in mercurial poisoning. Sometimes catarrhal stomatitis is developed by the spreading of an inflammation of the mucous membrane from the neighbouring parts, as, for instance, during the course of pharyngitis. Besides the purely traumatic influence of teeth which are cast off during the stomatitis that occurs in teething, we have to take into consideration the hyperæmia and inflammation of the gums (gingivitis). We have no positive knowledge of the possibility of stomatitis being developed from a chill.

**Symptoms.**—In all our domestic animals the symptoms of simple catarrhal stomatitis vary greatly according to the degree of the inflammation. In very slight cases we only find dryness and heat of the mucous membrane; but later on, a pappy, greyish-white, frothy coating appears, especially on the dorsum lingue. Such cases of slight stomatitis are most frequently observed as accessory symptoms of general febrile diseases. The so-called "coated" tongue is a result of an increased production of epithelium, owing to the catarrhal condition, and to the fact that the mechanical removal of the epithelium becomes checked, in consequence of the diminution in the amount of food consumed. It also occurs from the epithelial scales becoming dry and opaque on account of the reduced supply of saliva during the fever. The mucous membrane of the lips and cheeks may also become congested. The portion of the hard palate which is behind the incisor teeth is more or less swollen in consequence of the venous congestion of the very vascular submucosa, as in lampas of horses. There is also a mawkish, sweet smell from the mouth. In severe cases of catarrh there is increased redness and swelling of the mucous membrane, and especially of that of the lips, cheeks and gums. Also, an increased production of saliva and mucus causes viscid glairy strings to drip from the corners of the mouth; and more or less frequently, very abundant saliva is either beaten into froth, or flows in an uninterrupted stream from the lips (slobbering). The prehension of food is more or less suppressed.

If the epithelium be very abundantly cast off, the dorsum linguae becomes thickly coated, and assumes a dirty green, or brown colour, on account of its becoming soiled by particles of food; and a sweetish putrid smell issues from the mouth. In cats, the papillæ of the tongue, which resemble little horny teeth, appear strikingly white or yellowish white, in contrast to the bright red edges of the tongue. We sometimes observe in cattle and pigs hypertrophy of the epithelium of the inflamed cone-shaped papillæ, which has been erroneously regarded as thrush (the so-called "brush" on the tongue of cattle). In birds catarrhal stomatitis manifests itself by the horny covering of the tongue becoming dry and opaque, and consequently laymen often make the mistake of thinking it is a pathological membrane, and wrongly pull it off.

The erosions which occasionally occur during the course of simple catarrhal stomatitis, are small and superficial losses of substance of the mucous membrane, which usually heal rapidly, and therefore differ essentially from true necrotic ulcers. The same remark applies to the very rare ulceration of the mucous glands, which we have observed on a few occasions in horses during the course of strangles and rickets, and which were formerly known as *stomatitis folliculosa*. It should be regarded as a complication of catarrhal stomatitis. It begins with the formation of small grey or dun-coloured nodular elevations which are surrounded by a red area. The nodules appear as a consequence of the excretory ducts of the numerous glands becoming plugged up.

**Therapeutics.**—Catarrhal stomatitis is a very mild affection, which, as a rule, can be cured by the removal or exclusion of the irritating causes. A proper method of treatment greatly accelerates recovery, which is very desirable, because animals that are suffering from this disease do not feed well, consequently lose flesh and are incapable of doing hard work. The simplest treatment consists in the frequent irrigation of the mouth with fresh and pure spring water. Water acidulated with vinegar is an old domestic remedy for stomatitis. Such a mouth douche is generally prepared by adding a large tablespoonful of common salt and a quarter of a pint of vinegar to a quart of spring water. More obstinate cases may be treated with disinfecting solutions, such as 1 to 4 per cent. watery solutions of chlorate of potash or boric acid, and in severe cases,  $\frac{1}{2}$  to 1 per cent. of creolin, with which

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the mucous membrane may be irrigated or painted. Solutions of permanganate of potash are less reliable, because they are feeble disinfectants and have a very disagreeable taste. Astringent solutions—such as  $\frac{1}{2}$  to 1 per cent. of alum or tannin, decoctions of oak-bark, garden sage leaves (*salvia officinalis*), etc.—are indicated when there is a very large secretion of saliva and mucus. Internally we may, at the same time, give common salt, sodium bicarbonate, sodium sulphate, etc.

As regards feeding, we should bear in mind that in all severe cases of oral catarrh only fluid or soft food should be given. If unwholesome food is the cause of the disease, a change of diet should of course be made.

**Salivation (Ptyalism).**—Formerly salivation was regarded as an independent disease, but it is merely a symptom of various diseases. It is caused most frequently by the presence of stomatitis, and also by foreign bodies in the oral cavity, pharynx, and oesophagus ; by pharyngitis, and by obstruction in, or paralysis of, the organs of deglutition. Paralysis of the pharynx and oesophagus occurs in rabies, during gestation, in diseases of the brain, and poisoning, particularly by bunt (*Tilletia caries*), or other fungi in spoilt food. Deglutition is also generally impossible in tetanus. Salivation is observed, particularly with dogs, in diseases of the stomach and intestines, but seldom in helminthiasis. It appears in a few cases of parotitis, and, by reflex action, in diseases of the uterus and ovaries. Pilocarpine, arecoline, mercury, iodine and morphia are the chief drugs that excite salivation. The cause of salivation often remains obscure. For reducing the secretion of saliva, we may try atropine or hyoscyamine by injecting it subcutaneously in horses and cattle in  $\frac{1}{2}$  grain doses, and in  $\frac{1}{10}$  grain doses in dogs. Saliva has an alkaline reaction in health, but is found to be acid in various human diseases, such as pleuritis, encephalitis, rachitis, dyspepsia, and diabetes. In order to test the accuracy of this statement with respect to animals, we have made many experiments in cases of disease, but have never found an acid reaction of the saliva. The occurrence of an acid reaction of saliva in man may be accounted for by the formation of acid in the mouth caused by decomposing particles of food, hollow teeth, etc.

2. STOMATITIS APHTHOA (*vesicularis*). SPORADIC APHTHE OF THE ORAL MUCOUS MEMBRANE.

**Nature and Causes.**—In veterinary medicine the name of aphthæ is given to vesicles on the oral mucous membrane, which are produced by an accumulation of serum under the epithelium of the mucous membrane, and, consequently, stomatitis aphthosa is identical with vesicular stomatitis. A typical stomatitis aphthosa is found in foot-and-mouth disease, which is a specific infective disease that is discussed in Volume I. of this book. There is a contagious and enzootic aphthous stomatitis which appears occasionally in horses, cattle and sheep, and which is entirely independent of foot-and-mouth disease. It has been known from remote times as "sporadic aphthæ," and its causes are most probably fungi which attack forage, particularly those that infect Swedish clover (*wromyces*) and rape (*polydemus exitiosus*). These and other infective fungi produce in some cases a catarrhal, in other instances an aphthous, and sometimes even an ulcerous or croupy stomatitis. Also, the hairs of caterpillars and other substances may produce an aphthous stomatitis by intense irritation, or by individual pre-disposition. In some cases, the cause remains unknown; but, as a rule, it can be found in a changed condition of the food.

**Symptoms.**—The phenomena of aphthous stomatitis consist of the formation of vesicles on the mucous membrane of the lips, cheeks, tongue and gums, which vesicles are filled with clear serum, and are either isolated or massed in large numbers. They usually burst in a very short time and become changed into congested sores (erosions) which quickly heal. They vary in size from that of pin head to that of half a crown. Besides the vesicles, there are usually symptoms of catarrhal stomatitis, with redness and swelling of the mucous membrane and salivation. We may sometimes notice a "smacking" noise during the movements of the lower jaw, similar to that which takes place in foot-and-mouth disease. After the bursting of the vesicles, the mucous membrane of the mouth may remain painful for some time. There is failure of appetite, and simultaneously with the appearance of the vesicles, we may sometimes find a feverish rise of temperature, which rapidly subsides. It occasionally happens, particularly in cases

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of "clover disease," that, besides the inflammation of the mucous membrane, an erysipelatous dermatitis breaks out on the head udder, limbs and other parts of the body. The nature of the disease is generally benign and its course rapid.

**Differential Diagnosis.**—Sporadic aphthae may be readily confounded with foot-and-mouth disease. Experience teaches that a prompt and exact differentiation is not always possible, especially when several animals become simultaneously affected in consequence of the consumption of the same food, and when fever is also present, or when the ingestion of the fungi is followed by gastric symptoms, or by symptoms of a general mycotic intoxication. The fact that the disease produced by these fungi cannot be transmitted to healthy animals will be decisive, and, consequently, inoculation experiments should never be omitted in doubtful cases. Also, the coronet of the hoofs in sporadic aphthae is, as a rule, free from aphthous lesions, and the aphthous affection of the oral mucous membrane usually heals more rapidly than in foot-and-mouth disease, in which the vesicles are generally larger.

**Therapeutics.**—The treatment of aphthous stomatitis consists in the frequent rinsing out of the mouth with fresh spring water, avoidance of rough forage, and in the use of disinfectant and astringent lotions (boric acid, potassium chlorate, creolin, salicylic acid, and alum, tannin, sulphate of iron, etc.). Also, the mixture of vinegar, common salt and water, to which allusion was made (p. 4) when discussing catarrhal stomatitis, may be used as a domestic remedy.

### **3. STOMATITIS ULCEROSA.**

**Definition.**—Stomatitis ulcerosa consists in a necrotic inflammation of the oral mucous membrane, which spreads to a varying extent, and which attacks the gums more frequently than other parts. Anatomically we may regard it as a very severe form of the catarrhal and aphthous process, which manifests itself by the formation of deep ulcers on the mucous membrane.

**Etiology.**—Apart from traumatic, thermal and chemical causes, stomatitis ulcerosa is found most frequently in delicate, pampered, weak and anaemic dogs of the smaller breeds, and in cats. This disease is sometimes preceded by debilitating affec-

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tions, such as gastro-intestinal catarrh, rachitis, and distemper. Ulcers which appear in such cases were formerly looked upon as scurvy (an erroneous idea) because similar ulcers may be seen in otherwise healthy animals, without any haemorrhage into the mucous membrane, as in scurvy. The cause of ulcers in non-scorbutic stomatitis is probably abnormal vulnerability, and low power of resistance of the mucous membrane from individual idiosyncrasy, in combination with an infection. Carious teeth, or teeth which are thickly covered with tartar, are very often the starting point of the affection, on account of an infectious inflammation of the gums being set up by them. The statistics of the Berlin canine hospital have shown that stomatitis ulcerosa forms about one-half per cent. of all the cases of diseases of dogs at that institution.

Stomatitis ulcerosa is sometimes set up in horses, cattle and sheep by the consumption of food infected by fungi, chiefly pyrenomycetes and uredineae, which give rise to ulcerous inflammation of the mucous membrane. Thus stomatitis ulcerosa has occurred enzootically in horses from eating Swedish clover, and in lambs from feeding on infected rape-cakes. Certain bacteria have also been regarded as pathogenetic factors in this disease (Rivolta).

As a secondary symptom, we find ulcers of the oral mucous membrane in foot-and-mouth disease of ruminants and pigs; in stomatitis pustulosa contagiosa of horses; in avian diphtheria and in mercurial posioning.

**Symptoms in Dogs.**—The gums in the neighbourhood of certain teeth at first become dull red, a condition which is observed particularly near the incisors and canines, sometimes also round the molars, on both jaws, and on one or both sides; the portion of the gum surrounding the neck of the tooth is also swollen. Even in one or two days there is a well-marked increase in the swelling of the affected tissue, which is of a dark red, brown red, or purple colour, and looks spongy, swollen and loosened. It has receded a little from the respective teeth, and bleeds on the slightest pressure. Later on, the affected tissue becomes discoloured, this change being initiated by the swollen part which is next to the teeth, and which assumes a tint varying from greyish green to greyish yellow. As a consequence of the cellular infiltration and necrosis of the mucous membrane, we have the necrotic crust, which at first is friable, and, later on, greasy and pulpy. A spontaneous or

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forcible removal of this mass reveals a large ulcer, which is usually about one-sixteenth of an inch deep, of varying size, and sometimes circular when at the canine teeth. Its edges wind in and out in an irregular manner, are usually raised in the form of a wall, hard to the touch and very hyperæmic. The base of the ulcer is uneven and discoloured. At the same time, there is considerable slavering, and glairy, ropy, brown-red mucus, which is often mixed with blood, flows from the corners of the mouth, and soils all the adjoining parts. Such masses of mucus are also found in large quantities in the mouth itself, and are discharged when it is forcibly opened, a proceeding from which dogs generally try to escape, as it hurts them. The discharge, the entire oral cavity and the expired air all have a very foetid, nauseous and putrid smell, which often taints the entire neighbourhood of the patient. Besides the circumscribed localised ulcers, the symptoms of simple catarrhal stomatitis are also present.

The above described phenomena of inflammatory hyperæmia, swelling, cellular infiltration and necrotic disintegration of the mucous membrane appear not only on the gums in the immediate neighbourhood of the teeth, but also on other parts of the interior of the mouth, as, for instance, on the anterior surface of the gums, lips, corners of the mouth, less frequently on the cheeks, and very rarely on the lower surface of the tongue. The ulcers on the lips frequently occupy positions directly opposite those on the edges of the gums close to the teeth, and consequently we cannot help thinking that in such cases they arise from auto-infection. In other cases the arrangement of the ulcers suggests that infection was conveyed by the lymph passages. We also meet with instances in which the morbid process in no way affects the teeth or the mucous membrane in their immediate neighbourhood; but begins on other parts of the gums, on the lips, or even on the corners of the mouth, and occasionally spreads to other portions of the common integument.

**Process.**—In benign cases and in young animals, the ulcers enlarge only to a moderate depth and extent. The general health is but little disturbed, fever is absent or remains very low (about  $103^{\circ}$  F.), the appetite is surprisingly good, and after the ulcerative process has stopped and the ulcers have become clean, these lesions usually heal very quickly, even as soon as eight or ten days. Such cases are the most frequent.

In other instances, particularly with old animals that are suffering from another complaint, the infiltration and ulcerative disintegration of the affected tissue progress very rapidly. The ulcers become very deep and sinuous, the implicated teeth become loose and can be easily pulled out; their roots are covered with a grey, stinking, pulpy substance (the destroyed alveolar periosteum), and caries and necrosis may supervene. Sometimes a fistula is formed between the mouth and nasal passages, particularly on the site of the canine teeth, with subsequent suppurative rhinitis. The affected parts of the lips and cheeks are hard and tense to the touch, are more or less oedematously swollen, and there is an inflammatory swelling of the adjoining lymph glands. In these cases the general health is more or less disturbed, and recovery may not take place for several weeks or even months.

If the course of the disease be unfavourable, we may often observe, during freshly appearing eruptions, that persistent fever of a varying character sets in; the pulse becomes more and more frequent and smaller, the appetite is entirely lost, diarrhoea makes its appearance, the animal becomes very weak and prostrated, and finally dies from septicaemia.

**Symptoms in Herbivora.**—When stomatitis ulcerosa of horses, cattle and sheep is produced by fungi, it manifests itself by ulceration, swelling, congestion and pain in the mucous membrane of the mouth and lips, and by copious slavering. In severe cases, the oral mucous membrane, especially of the gums, palate and sides of the tongue, becomes affected with circumscribed, diphtheria-like changes in the form of grey or grey-brown prominent spots, which vary in size from a pea to half a crown, and which, after removal of the crust or core, show a deep red, and sometimes a briskly granulating base. We may not unfrequently notice severe general disturbance (mycotic poisoning), which is specially well marked in young animals, and which has been described by Berndt in connection with lambs. He found that the principal symptom was an acute inflammation of the nose and mouth, which almost always ended in death. The general symptoms consisted in great debility, staggering gait, accelerated and difficult breathing, and faint short cough. The mucous membrane of the gums and the tongue showed numerous erosions, which were also present on the visible part of the Schneiderian membrane, where they were covered by a thick brown crust. On *post mortem* examination, the mucous membrane of the nose, mouth, pharynx and larynx were found swollen and partly eroded; the lower edges of the posterior lobes of the lungs were hard and dark red; their section-surfaces, from which an ichorous fluid flowed, were granulated; and the divided bronchi contained a frothy fluid, which could easily be pressed out. Microscopic examination showed that numerous brown, ramified threads and chains of spores of *polydesmus excisus* existed between the epithelial cells of the oral and nasal mucous membranes. These lambs had

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been fed on rape cakes, in which spores of the ascomycetes were found. After the feeding on these cakes was stopped, no more cases of infection occurred. Rivotta has described the occurrence, in young lambs, of a similar very malignant epizootic stomatitis ulcerosa, which was complicated with conjunctivitis, pneumonia and hepatitis. Gips, Bertsche and others have reported similar cases of stomatitis in lambs.

**Therapeutics.**—The treatment of stomatitis in dogs is essentially surgical, and consists in careful and repeated disinfection of the affected mucous membrane. As mild antiseptics, such as boric acid, salicylic acid and potassium chlorate, are not generally effective, we would recommend, from the very first, the application of the strongest disinfecting agents, of which creolin is one of the best. By the use of a 1 to 2 per cent. watery solution of creolin we have obtained surprisingly good and rapid results. The solution of creolin should be carefully applied with a brush four or five times a day to each ulcer. A 1 to 4 per cent. solution of aluminium aceto-tartrate or zinc chloride; solutions of perchloride of mercury (1 to 1,000), sulphate of copper and nitrate of silver (1 to 2 per cent.); and touching the ulcers with caustics are very efficacious means. When using the last-mentioned metallic salts, great care should be taken that only the ulcers are touched, and that none of the fluid is swallowed. We have sometimes observed favourable results from the application, with a brush, of alcoholic remedies, such as tincture of myrrh and aloes. The early and free extraction of carious and loose teeth is of essential importance for recovery.

### **PAROTITIS (*Inflammation of the Parotid Gland*).**

**Occurrence.**—Although this disease is comparatively frequent among mankind, in whom it is commonly known as "mumps," it has received but little attention in veterinary literature, which neglect is explained by the fact that it very seldom occurs among our domestic animals. It is particularly rare in dogs. Out of 70,000 cases of sick dogs in the Berlin hospital, during nine years, there were only six cases of parotitis.

**Etiology.**—This disease may be traced to several causes:—

1. *Traumatic parotitis*, produced by mechanical injuries (should be treated surgically).
2. *Primary idiopathic parotitis*, which is caused by a mias-

matic contagium, and which appears epizootically. To this form we may ascribe the cases observed by Aruch in horses, by Franze in goats, by Hertwig in dogs, and by Schüssele in dogs and cats, which were epizootic and analogous to epidemic mumps in man. As regards the pathogenesis of this chief form of parotitis, it is possible that the contagium is carried by the blood to the parotid gland, or, what is more probable, that it reaches it from the oral mucous membrane *via* Stenon's duct.

3. *Parotitis caused by the extension of a neighbouring inflammatory focus*, as, for instance, by the presence of an impacted salivary calculus, or by inflammation of the buccal membrane spreading through the salivary duct, as in a case described by Toussaint, in which the inflamed salivary duct became dilated into the shape of a funnel. The morbid process in strangles, angina and distemper, may extend to the parotid gland in a similar way.

4. *Metastatic parotitis* during the course of pyæmia and septicæmia, and, as stated, in strangles, pleuro-pneumonia and distemper.

5. *Parotitis caused by the immigration of actinomycetes bovis* (*Parotitis—actinomycosis*) is a secondary symptom of actinomycosis, which is an infective disease, and occurs epizootically among cattle in certain districts. This parotitis differs from the other forms by being chronic.

Stockfleth describes, under the heading of "Chronic Inflammation of the Salivary Glands," in his book on surgery, a disease which is without doubt actinomycotic parotitis. He explains that the inflammation occurs particularly in cattle, which graze on low-lying meadows near the sea-shore, and are fed with hay produced on these meadows. The parotid gland is much more frequently affected than the submaxillary or sublingual. The course of the disease is very slow. The gland shows hard, uneven, painless swellings, which increase in size very gradually, and usually form abscesses later on. A secondary swelling of the lymph glands nearly always supervenes. Finally, the swollen gland presses on the upper part of the pharynx and larynx and causes difficulty in deglutition and respiration. The administration of medicines is entirely useless, and a cure can be expected only by excision of the swollen gland. For further information on actinomycosis and glossitis, see p. 204, Vol. I.

**Symptoms.**—In the three forms of parotitis (idiopathic, metastatic, and the form that spreads from a neighbouring focus) which we have to deal with here, there is a more or less well-marked swelling of the parotid gland. In idiopathic parotitis this swelling spreads more uniformly over the gland than in the other two varieties, and seldom suppurates. In the other two

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forms it is more circumscribed, and is usually followed by suppuration of the implicated lobules. It may also spread to adjoining parts. Thus, Franz observed in an epizootic attack of this disease in goats, that the upper and lower jaw and the submaxillary glands were swollen and inflamed, and consequently the animals were unable to eat or drink. Aruch saw during eighteen months only one unilateral case among seventy horses which were suffering from parotitis and maxillitis. At the same time, there were symptoms of stomatitis. We have no exact knowledge about the other symptoms of parotitis, and are particularly ignorant with respect to the course of its fever. We have never observed salivation during parotitis.

**Differential Diagnosis.**—Parotitis has probably been confounded most frequently with inflammation of the sub-parotid lymph glands during strangles and pharyngitis, which mistake could certainly be avoided by a knowledge of the anatomical position of these lymph glands, and of the history of the case. We may also have to consider swelling of the guttural pouches, affection of veins caused by a fistula produced by phlebotomy, simple edematous or phlegmonous inflammation of the skin and, especially, of the subcutaneous connective tissue which covers the parotid gland, and various kinds of swellings of the parotid gland.

**Therapeutics.**—The treatment of non-suppurating parotitis consists in the application of moist heat (Priessnitz's bandages) and inunction with ointment of camphor, creolin, carbolic acid, or iodoform. In chronic cases more active means, such as tincture of iodine, biniodide of mercury ointment and cantharides ointment may be tried. For actinomycotic parotitis of cattle, we would recommend the internal administration of potassium iodide, from 1 to 4 drams daily (or what is equally effectual and far less expensive, from 5 to 7 grains of biniodide of mercury, dissolved in water by means of an equal quantity of potassium iodide). Abscesses should be opened as early as practicable. Any resulting salivary fistulae should be treated surgically.

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**General Remarks.**—The name of angina (inflammation of the throat) has for a long time been applied generally to inflammation of the mucous membrane of the pharyngeal

cavity and its adjoining parts, especially the larynx; because an exclusive affection of the pharynx is rather rare, particularly in horses. Sometimes pharyngitis is best marked in this inflammation of the throat; at other times, laryngitis. Hence it is advisable to divide this complaint into pharynx-angina and larynx-angina, according as pharyngitis or laryngitis is more in evidence. We shall here discuss only pharynx-angina, and will put larynx-angina among the "Diseases of Respiration" (p. 650).

In the smaller domestic animals we are able to sub-divide pharynx-angina into separate local affections, according to its respective sites on different parts of the pharynx, as, for instance, inflammation of the soft palate, tonsils, and true pharynx; but in large animals, like horses and cattle, we cannot do this without complicated instruments. The term pharynx-angina, therefore, signifies in veterinary medicine, a more or less extensive inflammation of the mucous membrane of the pharyngeal cavity, including the soft palate and tonsils, in which case, usually, though in a subordinate manner, the mucous membrane of the larynx, upper part of the trachea, mouth, nose and rarely that of the Eustachian tubes and guttural pouches become involved.

The difficulty of clinical examination of the larger animals makes it hard to find out the character and form of the disease. The differentiation of catarrhal, phlegmonous, suppurative, haemorrhagic, croupy, diphtheritic, lacunous and necrotic forms can be easily made only in the smaller animals. We can, however, make a fairly exact examination of the mucous membrane of the pharynx of horses by means of Polansky and Schindelka's rhino-laryngoscope, which is too complicated for general practice, and cannot be used with cattle. We can, therefore, as a rule, only indirectly infer the presence of one of the above-mentioned forms of pharyngitis, as, for instance, the catarrhal or phlegmonous variety, from the difference in the intensity of the symptoms, secondary implication of the lymph glands, fever, duration of the disease, etc. The croupy form can be recognized only by the ejected or coughed-up croupy membranes, and even in this case we are not able to decide whether the croupy shreds come from the larynx, or from the pharynx. We shall, therefore, try to give a general sketch of pharyngitis, without laying stress on the classification of the above-mentioned special forms.

**Etiology.**—Pharyngitis occurs sporadically or epizootically in all the domestic animals, but most frequently in horses, dogs and pigs; after them, in cattle and cats; and most rarely in sheep and birds, except in cases of avian diphtheria. Among general causes, we may assume the influence of individual

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predisposition in the domestic animals, although it is not so well marked as in man. The true nature of this predisposition in both animals and man is unknown. It is, however, certain that young animals and those which are weak, pampered, and habituated to warm abodes, frequently suffer from this disease, although old and robust ones are not entirely immune. The following are the chief special causes of pharyngitis:—

1.—Mechanical, chemical and thermal irritants which directly affect the mucous membrane of the pharynx. Our injuries thus produced, we may include bruises and lacerations of the mucous membrane caused by sharp meat-bones, fish-bones, thorns, needles, rough, coarse and prickly food, awns of barley, careless manipulation of the oral cavity (as, for instance, skilful filing of the teeth), etc. Larvæ of gad-flies which have attached themselves to the mucous membrane of the pharynx, may produce a more or less chronic pharyngitis. Also tumours that project into the pharyngeal cavity may mechanically cause catarrhal inflammation of the mucous membrane, as we find in the case of actinomycetoma and tubercular new-growths, both of which are common ailments in cattle. The chief chemical irritants are the poisonous plants mentioned in connection with catarrhal stomatitis (p. 2); drugs which, usually from inaccurate compounding, cause erosions of the mucous membrane, as, for instance, tartar emetic in an undissolved condition, croton oil in too concentrated solutions, ammonia not sufficiently diluted, metallic salts, arsenic, phosphorus, etc. Fodder which contains pyrenomyces and uredineæ sometimes give rise to both stomatitis and pharyngitis. Hot drinks which scald the mucous membrane, hot food, and the inhalation of steam may also set up angina.

2.—Chills, especially local chills in the pharynx, caused by the inhalation of cold air when north or north-east winds prevail, by the drinking of very cold water, the consumption of food covered with hoar frost, etc., may act as local irritants in setting up pharyngitis. We have also to consider the effect of cold in a wider sense, as, for instance, chill of the surface of the whole body, in consequence of a sudden change of weather, rain, or any great fall of temperature. Although it is certain that too much importance has been attached to the influence of chill in the production of disease, yet with our present state of knowledge this factor cannot be entirely ignored, especially in the case of pharyngitis.

3.—*The spreading of inflammation from neighbouring organs*

*to the mucous membrane of the pharynx.* Stomatitis, as well as rhinitis and laryngitis, may cause pharyngitis by spreading. As pharynx-angina is the most common symptom in strangles, the two diseases are often mistaken for one another. It also occurs as a complication in foot-and-mouth disease, rabies, distemper, pleuro-pneumonia, petechial fever, anthrax, acute and chronic glanders, tuberculosis, etc.

4.—Infection. Many cases of pharynx-angina appear to be caused by a specific infection, by which the disease assumes a contagious character. Outbreaks of epizootic angina certainly bear out this supposition. In this respect we must be very careful, especially with horses. The fact that strangles frequently exhibits symptoms of pharyngitis, has certainly caused many mistakes to be made between this disease and epizootic strangles. It is wrong always to regard equine epizootic pharyngitis, accompanied by suppuration of the glands, as strangles. Infectious epizootic pharyngitis, which may lead to metastatic pyæmia, sometimes occurs among puppies during the first few weeks after birth.

The attachment of bots on the pharyngeal mucous membrane must not be accepted off-hand as a cause of pharyngitis, because we know from experience that these larvae are frequently found on this membrane in perfectly healthy horses, as they also are in the stomach. The case of fatal pharyngitis in a remount, described by Lichmann, in which 14 bots were found on the pharyngeal mucous membrane, cannot be referred to these larvae; because the animal was one of several horses which were suffering from severe inflammation of the throat.

**Pathological Anatomy.**—In mild cases of catarrhal pharyngitis the mucous membrane of the pharynx is either diffusely congested or shows red spots; it is swollen; and covered with glairy or purulent mucus, which may be streaked with blood. It sometimes shows long narrow erosions. In chronic cases the mucous membrane is pale, thickened, wrinkled and covered with thick, ropy mucus. The swollen follicles and mucous glands project above the level of the mucous membrane in the form of nodules, which are about the size of a grain of millet.

In severe cases (*pharyngitis parenchymatosa et purulenta*), the highly congested mucous membrane either becomes œdematosely swollen, in consequence of a serous infiltration, and may even present a glassy and gelatinous condition; or it appears as if scalded, and is covered by a purulent, or dirty ichorous, necrotic, or greenish greasy coating, under which

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losses of substance to a various extent are found, and consequently the mucous membrane frequently presents a, so-to-speak, "gnawed" aspect. The lymph follicles and mucous glands are swollen to the size of peas, or are suppurating. Incisions into the mucous membrane, particularly in the region of the tonsils, often show numerous plugs of pus, which may become partly changed into concretions of lime.

If the affection of the mucous membrane spread to the submucosa, it will assume a more phlegmonous character (*pharyngitis phlegmonosa*), in which case the submucosa becomes infiltrated with a serous fluid or with haemorrhages, and is often changed into a gelatinous or purulent substance. In this way, abscesses of various size form under the mucous membrane, lift it up from its base, cause it to become bulged and perforated, or render it gangrenous by the tension they exert on it. The purulent, phlegmonous breaking down of the submucosa may proceed to a wide extent along the loose connective tissue, as, for instance, from the pharynx at the posterior surface of the oesophagus and of the trachea, to the entrance into the chest. This process gives rise to new abscesses and long fistulae; the mucous membrane often throws off shreds of dead tissue and becomes ichorous; and particles of food, which maintain and increase the suppuration and necrosis, accumulate under the detached mucous membrane. In one case of this kind, Bruchmüller saw the mucous membrane of the entire oesophagus, loosened by the penetration of food.

The retropharyngeal lymph glands, upper cervical glands, and sometimes even the submaxillary and sublingual lymph glands also simultaneously participate in the inflammatory process. These lymph glands in the neighbourhood of the pharynx and larynx become swollen and haemorrhagically infiltrated, and form abscesses or become disintegrated into a dark red, pulpy mass. The so-called retropharyngeal abscesses usually contain thick, white, creamy pus, and are either simple or winding. They may contain a fifth of a pint of pus or more. Later on the pus often turns caseous, and becomes encapsulated by a horny hyperplasia of the surrounding connective tissue. In severe cases, the submaxillary and sublingual lymph glands suppurate, and give rise to fistulae which open outwards.

The mucous membrane of the larynx is frequently congested, diffusely or in spots, and infiltrated in some parts with

serous fluid (œdema of the glottis); the epiglottis is œdematosily swollen at its base, and the guttural pouch sometimes contains thickened masses of pus. This filling of the guttural pouch does not occur as often as it is supposed to do; for in many cases sub-parotid and retropharyngeal abscesses are evidently mistaken for it, as we may see when they are surgically opened. The mucous membrane of the Eustachian tube often becomes thickened. The bronchi also suffer from catarrhal changes, and sometimes they become plugged with pus, and their mucous membrane becomes ulcerated, with the result that the cartilage is laid bare. The lungs often show circumscribed pneumonic foci with necrosis (pneumonia due to foreign bodies), and are often filled with stinking ichorous cavities of the size of a pigeon's egg, in the neighbourhood of which pleuritic coatings form. The Schneiderian membrane is usually excessively congested (either brick red or discoloured), covered with clots of blood or pus, which may assume a croupy appearance, and which sometimes can only with difficulty be detached. We may also find the under part of the mucous membrane here and there velvety or villous, and the villi provided with blood-filled vascular loops. The oral mucous membrane is often catarrhal, which condition may spread to the intestines. We sometimes meet with a desquamative inflammation of the mucous membrane of the rectum.

In a protracted course of the disease, the *heart* shows a parenchymatous and fatty degeneration; and the *liver* exhibits swelling and fatty degeneration. These symptoms, so far from being peculiar to pharynx-angina, are common to all diseases which are accompanied by high fever and great exhaustion.

With respect to the anatomy of the pharyngeal mucous membrane when in a normal condition, the following points are of importance for the pathology of pharyngitis. The mucous membrane of the anterior surface of the velum palati contains, according to Franck, a thick layer of yellowish, acinose, mixed glands, namely, mucous glands and serous glands (Ellenberger's palatine glands), interspersed among which we find (only in pigs) numerous small lymph glands. The surface of the pharyngeal cavity possesses, on the contrary, only a very thin layer of these mixed glands. At the base of the tongue near the tonsils we find the so-called follicular glands, which represent the lymph glands of the tongue. The dorsal part of the pharynx contains many lymph follicles, along with numerous mucous glands on its upper edge, and particularly in the neighbourhood of the orifices of the Eustachian tubes. It is noteworthy that

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in horses and pigs the tonsils, which lie in the posterior part of the oral cavity, possess many *foramina coeca*, whilst in the other domestic animals there is only a single deep fossa, which fact may perhaps partly account for the frequency of angina in horses and swine.

**Symptoms in Horses.**—I. *Pharyngeal symptoms.* A veterinary surgeon has seldom the chance of observing the beginning of the disease. Its development is usually so slow, that for the first three or four days, or even longer, the owner does not as a rule notice that the animal is ill, until his attention is called to that fact by its gradual loss of appetite and by the appearance of a cough, which are the preliminary symptoms of many other diseases. Consequently, even a qualified man is often unable to make an exact early diagnosis of this ailment. In a short time, however, characteristic symptoms appear. First of all, there is the peculiar and well-marked stiff carriage of the head, which is a consequence of the effort made by the patient to avoid any unnecessary movement of the head and neck. In this case the animal often manifests an extraordinary sensitiveness to pressure on the region of the larynx and pharynx. If we watch him while he is feeding, we shall see that although appetite is present, mastication and deglutition are more or less difficult, and swallowing is always very painful, and performed with much exertion, while the head is kept stretched out in a marked manner. Sometimes the function of deglutition is entirely suspended. The difficulty of chewing and swallowing leads to an accumulation in the oral cavity of mucus and saliva, which, when the mouth is opened, are emitted in the form of long transparent strings, and soil the parts adjoining the lips, the manger, and other objects within reach. These retained secretions are usually abnormally hot and have a mawkish smell, in consequence of the septic decomposition of the cast-off epithelium and fluids. After the fodder has been masticated for some time, it becomes covered with tenacious, glairy mucus; is worked up with saliva into a froth, and then ejected in the form of smooth balls or cakes, in which case particles of food may be left in the lateral divisions of the oral cavity. In these circumstances the patient instinctively prefers soft or sloppy to dry food. A portion of the water that is taken into the mouth is discharged through the nostrils, in which case, it washes out the tenacious mucous masses which have accumulated in the nasal passages. Also masticated masses of fodder are ejected through the nostrils, and frequently

give a green colour to the nasal discharge, by means of the chlorophyl they contain. This so-called "regurgitation" is the most important diagnostic symptom of pharyngitis. It arises from the fact that the muscles of the palate and pharynx are unable to act, on account of their suffering from serous infiltration or from the inflammatory process having spread to them.

It is evident from this explanation, that the more violent the regurgitation, the deeper and more phlegmonous is the pharyngitis. As even the swallowing of fluid causes pain, the patient, as a rule, does not drink much, but likes to play with the water put before him. In some animals we may now and then notice a painful effort at deglutition being made, without anything being swallowed. The oral mucous membrane generally shows symptoms of catarrhal stomatitis, namely, high temperature, congestion, coating of the tongue, etc. An inspection of the pharynx (when practicable, as in dogs and cats, or with the rhino-laryngoscope, for horses) discloses hyperæmia, swelling, haemorrhages, erosions, and, in fact, all the changes already described (p. 14).

2. *Respiratory symptoms.* The Schneiderian membrane generally manifests catarrh, and is often affected by intense congestion, particularly well marked on the lower edge of the posterior nasal passage. The nostrils discharge a fluid which at first is serous, or appears as a fine froth, but later on is muco-purulent, assumes a white, grey, yellow, or even green colour, and dries into a greasy crust on the *ala nasi*. Cough is almost always present, and at first is dry, sometimes "barking," but sputum is soon thrown up. The cough is generally very painful, on which account the animal tries to suppress it. It comes on in paroxysms, during which the mucous masses, etc., that have accumulated in the mouth, are discharged. The cough is caused partly by the spreading of the inflammation to the mucous membrane of the larynx, and partly to irritation of this membrane by the presence of unswallowed secretions which are kept back in the pharynx. The cough can often be extremely easily excited by pressure of the larynx. In some cases it is brought on by any forcible movement of the head. Respiration is disturbed in proportion to the implication of the larynx, the intensity of the fever, etc. In sight cases the respiration is very little accelerated, although it is generally difficult and wheezing, and may be even stertorous and rattling. We can hear various moist

## PHARYNGITIS AND PHARYNX-ANGINA.

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rales in the larynx and trachea, on which we can feel the vibrations caused by the ingress and egress of the air (*fremitus*).

3. The parts adjoining the pharynx, and especially the lower part of the parotid region, are frequently swollen and hot. The swelling is usually bilateral, but, as a rule, is not equally intense on both sides. It is either flat and diffuse, so that the normal outlines become somewhat indistinct, or it is sharply defined; although it may become exceedingly extensive and fill out the entire space between the lower jaw and the atlas. In most cases it is the lower part of the parotid gland which becomes prominently raised by the inflammatory infiltration of the lymph glands that lie under it. The supporting tissue of the parotid gland may participate in the swelling, and also the guttural pouches in exceptional cases. Bell has seen an instance of empyema of the guttural pouches, with temporary deafness, during the course of an attack of pharyngitis in a horse. Also the skin in the neighbourhood of the larynx may become swollen in consequence of collateral oedema. Besides, we may find tumefaction of the glands of the intermaxillary space, submaxillary, sublingual, and sometimes of the thyroid gland. The swellings in the parotid region are generally very painful.

4. The *digestive apparatus* frequently shows symptoms of severe intestinal catarrh; the patient often yawns; peristaltic action is more or less suppressed, and may become entirely arrested; the faeces are stinking, broken up, soft and covered with mucus, and the urine is acid. If there be no intestinal catarrh, and no great diminution of appetite, the urine will retain its alkaline reaction, despite the fact that high fever may have existed for weeks. In severe cases the urine contains albumen. A very low specific gravity of the urine is a striking pathological sign which is sometimes present. Thus we have observed cases in which specific gravity of the copiously discharged urine varied during the three days from 1001 to 1006 (normal s.p. 1014 to 1037); the specific gravity appearing to be dependent on the severity of the disease.

5. *Fever.* The nature and mode of appearance of the fever of pharyngitis is by no means typical. It may be absent in mild cases, or the pulse may show only a slight acceleration, as for instance, up to 48 beats in a minute. In other instances the disease sets in with high fever; the temperature being as high as 104° F., or 106° F., or even higher, while

the pulse for the first few days is little, if at all, increased in frequency. We have cases in which, with a temperature of over  $104^{\circ}$  F., the pulse did not exceed 40 beats a minute during the whole course of the disease. As a rule the pulse gradually follows the temperature, as long as the action of the heart is influenced by the prolonged effect of such high fever. If the disease begins on the very first day with high fever, we must conclude, as in other diseases, that it has been caused by infection. Pharyngitis, which appears during the course of strangles, is also distinguished by high fever. A gradual rise of temperature to a considerable height points to an intense inflammatory process, especially of a phlegmonous nature. A sudden rise of temperature is usually connected with the formation of abscesses in the lymph glands, etc. A marked rise in the frequency of the pulse is always a bad sign. In the horse, 80 pulsations in a minute are ominous; but a high degree of temperature is not so serious. High fever manifests itself not only by the frequency of the pulse and the temperature of the rectum, but also by unequal distribution of heat in the surface of the body, as for instance, cold ears and cold extremities, with considerable injection of the conjunctiva.

6. During exceptional cases of pharyngitis, we have observed intermittent eruptions of pomphi, which appear and disappear suddenly. This condition probably indicates a vaso-neurotic or embolic exanthema, as is occasionally the case in other infective diseases, for instance, strangles.

**Process.**—In the great majority of cases the course of pharyngitis is acute, although certain sequelæ and complications may protract the duration of the disease. True chronic pharyngitis is found only when the action of the exciting irritants is long continued, as in the case of bots or tumours in the pharynx. As a rule, the disease ends in resolution, particularly in cases of simple catarrhal angina, which recovers in from six to fourteen days. On the other hand, cases of pharyngitis in which there is phlegmonous inflammation of the submucosa, or suppurative infiltration of the retropharyngeal and sub-parotid lymph glands, are of longer duration. The abscesses which form in such cases produce, by the size of the collateral swelling in their neighbourhood, difficulty of breathing and increase of temperature, and may thus protract recovery for four or five weeks, or longer, or may cause death by suffocation.

cation, in consequence of a rapidly forming oedema of the glottis. These abscesses rarely open outwards; but, probably more often than is thought, break inwards by necrosis of the pharyngeal mucous membrane, and their contents are then discharged through the nasal and oral cavities, when the dyspnoea, as a rule, will be suddenly relieved; or they may run into the larynx and trachea, in which case the patient may be suffocated. Sometimes a necrotic inflammation of the guttural pouches, hyoid bone, and even of the occipito-atloid articulation may become developed (Ridge). In very rare cases an abscess opens both inward and outwardly, and thus produces a pharyngeal fistula, as Romant observed in a foal, out of which fistula milk escaped while the foal was suckling. We have seen two similar cases in horses. In one of these, after the abscess, which was the size of a man's fist and at a depth of four inches in the region of the parotid gland, had been surgically opened, it burst spontaneously inwards. In the other case there remained after recovery a running fistula, through which fluid could be injected into the pharynx and mouth. Parts affected by burrowing and perforating suppuration may become ichorous, which is a condition of great danger, as it may lead to septic poisoning, in which case the temperature is extremely high, the breath has an intensely putrid smell, and the animal coughs up necrotic, foetid shreds of tissue.

If the inflammatory infiltration of the glands does not lead to the formation of abscesses, and to the outward discharge of their contents, the glands are liable to become indurated, caseous and calcified, which condition causes a permanent increase in the circumference of the region of the larynx and pharynx, and thereby produces more or less difficulty in breathing. Paralysis of the pharynx has been observed as a sequel of pharyngitis.

Inflammation of the lungs is a very dangerous, and by no means rare, complication of phlegmonous and necrotic pharyngitis. It is the most frequent cause of death. As in severe cases the possibility of this complication is always present, it is the imperative duty of the veterinary surgeon carefully to examine the chest during the course of the disease. This inflammation of the lungs is in most cases due to foreign bodies, and is caused by portions of the oral contents having passed down the trachea. It is characterised by rapid gangrenous destruction of the affected parts of the lungs, and may be produced by forcible or unskillful drenching, or by emboli, which

arise from the ichorous foci of the necrotic pharyngitis (metastatic pneumonia).

**Differential Diagnosis.**—It is easy to diagnose a developed and uncomplicated case of pharyngitis in the horse, but is often hard to distinguish it in its early and later stages from other affections which have similar symptoms. Thus, confusion may occur in cases of foreign bodies being lodged in the mouth, pharynx and oesophagus, and with new growths in the pharynx, particularly polypi, actinomycoma and sarcomata, which render deglutition difficult. Affections of the pharynx, such as stenosis, paralysis and spasms of the part, often have similar symptoms; and parotitis, glossitis, stomatitis, swelling of the guttural pouches, ptyalism, etc., may lead to confusion. At the beginning of this chapter we have alluded to strangles, which of all complaints can be most easily mistaken for pharyngitis.

**Prognosis.**—Prognosis depends on the intensity and extent of the affection and on the complications that are present. Although a mild attack of catarrhal pharyngitis quickly recovers, the prognosis of a somewhat severe case should always be made with reservation, because sudden and unexpected death may ensue from gangrene of the lungs, or oedema of the glottis. Much also depends on whether or not the inflammation has spread to the larynx.

**Therapeutics.**—In an enzootic outbreak the affected horses should be segregated as a prophylactic measure. The stable should be of medium temperature, well ventilated, but free from draughts, and the food easy of mastication and deglutition. It is consequently advisable to give, if practicable, only green food, or very soft hay, or perhaps mashes of meal and bran. Oats and chop should be entirely avoided. Care should be taken that fresh water is kept constantly before the horse. The lips and edges of the nostrils, which get covered with mucus and crusts should be carefully cleaned, and the mouth frequently washed out with cold water or with some mouth douche, such as solutions of salt and vinegar, potassium chlorate, etc. If the mouth be rinsed out with a syringe, this instrument should be used very carefully, and the jet be directed towards the cheeks, and not in the direction of the

pharynx. The surroundings of the patient should also be cleansed.

In severe phlegmonous processes accompanied by great pain, tension and heat, it is advisable in the beginning of the attack to put cold applications or even ice round the throat ; and later on, when the abscesses begin to form, or even when the swellings are of a less inflammatory character, Priesanitz's bandages are of the greatest use, especially if the inner layers of the bandage are moistened with carbolised water instead of ordinary water. These bandages hasten the ripening of the abscesses and soften the hard inflammatory swellings.

The inunction of grey mercurial ointment on the swollen parotid region is much practised ; but it is doubtful whether sinapisms and stimulating ointments applied to the region of the parotid gland and intermaxillary space are of any use. They certainly should not be indiscriminately used. All that can be said for them is, that they hasten the maturing of the abscess more than moist heat. There is no ground for the idea that they have a derivative effect from without. They might be used with advantage on very circumscribed hard swellings, which ought to be quickly softened. On the other hand, these severe inunctions should not be used in cases of widely extended swellings, severe local inflammation, deep and diffuse suppuration, impaired action of the heart, or general debility, nor with languid young animals of weak constitution, because their application may give rise to enormous swelling, considerable dyspnoea and rapid gangrenous destruction of the cellular tissue, in consequence of which many horses have died.

After we have ascertained by palpation the presence of an abscess, we should not lose much time with blistering ointments, but should open it as early as practicable by cutting the skin, and then penetrating downwards with the fingers in a preparatory manner. We may thus open abscesses at a depth of four inches. Their further treatment is a matter of surgery.

Inhalations of steam, either plain or medicated with carbolic acid or creolin are much used ; but we should avoid having them too hot. Moreover, their value is probably insignificant. The local employment of astringents is of more importance, although it cannot be easily practised on horses. Kagel advises the injection of medicines into the pharynx by introducing an india-rubber tube into one of the nostrils. The tube used for this purpose is about two feet long, has a thick wall, and small calibre. About 3 oz. of the fluid is used, and it consists

of  $\frac{1}{2}$  per cent. solution of common salt, to  $\frac{1}{10}$  per cent. of corrosive sublimate, etc. The astringent or disinfecting solutions ( $\frac{1}{2}$  per cent. of nitrate of silver, Lugol's solution, 1 per cent. of lactic acid,  $\frac{1}{2}$  to 1 per cent. of alum) recommended by Dieckerhoff for intrapharyngeal injections, sometimes render good service; but are not always without danger, as we can state from our own experience.

We should be cautious in administering drugs internally, and under all circumstances fluid medicines should be avoided. Electuaries and balls should be used only to a small extent. Fever and constipation are the only conditions that require internal medicines, and we should try to combat them by frequent enemas of solutions of common salt and of soap and water, and by subcutaneous injections of appropriate remedies, as for instance, physostigmine for constipation, if dyspnoea be not present, or a solution of tartar emetic, for instance, in the drinking water. Pilocarpine and arecoline, are contra-indicated in pharyngitis, because these aperients produce at the same time copious salivation, which may become dangerous if the power of swallowing be impaired. The high fever of pharyngitis often resists the action of febrifuges, and we have frequently to fall back upon cold enemas and cold packing of the trunk. Antipyrin is the best febrifuge, because it can be subcutaneously used. It is injected in doses of from 150 to 300 grains dissolved in equal parts of water. In septic fever subcutaneous injections of spirits of camphor (150 to 300 grains, hourly) are indicated.

It may be necessary to perform tracheotomy in cases of pneumonia following on pharynx-angina, or hyovertebrotomy in order to empty distended guttural pouches. As success from tracheotomy depends upon its being performed at the proper moment (we cannot expect a real benefit from it, if we wait too long), and as the operation may become necessary at any moment, the instruments for its performance must be kept in readiness. Pneumonia due to foreign bodies, or metastatic pneumonia is to be treated according to general principles. The treatment, however, is often unsuccessful.

**Pharyngitis of other Domestic Animals.**—There is so great uniformity in the chief symptoms of pharyngitis, that a detailed description of the disease in our various domestic animals is superfluous. With regard to cattle, we may refer to the morbid conditions in horses. The chapter on actinomycosis may be consulted in the treatment of this disease. In the following remarks we shall discuss pharyngitis, particularly in pigs and dogs :—

1. Pharyngitis of pigs, which frequently occurs, has been fully

described by Spinola in his "Diseases of Pigs." In swine, as in other animals, it is easily confused with larynx-angina. We must also distinguish between pure idiopathic angina and anthrax-angina. Swelling of the throat is one of the most prominent symptoms in anthrax in swine. According to Spinola, the principal cause of pharyngitis is the drinking of very cold water when the body is heated. On which account the disease is particularly prevalent during spring in mountainous districts among pigs which graze near mountain streams. We have also to consider mechanical and chemical causes.

The symptoms of pharyngitis in swine vary according to the severity of the disease. In slight cases there is little or no fever. We chiefly find congestion of the visible mucous membranes, frequently lachrymation, accelerated and more or less difficult breathing, dry cough, hoarseness, slightly increased temperature of the mouth, difficult deglutition, retching, vomiting and swelling and congestion of the pharynx, which is hot and painful. Although the appetite is impaired, it is seldom suppressed. The disease runs its course in from ten to fourteen days, when the cough gradually becomes moist, and recovery as a rule takes place. According to Spinola, the symptoms of severe inflammation are: considerable congestion and dryness of the snout and visible mucous membranes, especially that of the mouth; shining eyes; staring look; the region of the pharynx is congested, abnormally warm, swollen and painful; high temperature of the mouth; oppressed and accelerated breathing, which in severe cases is very difficult; indistinct, short and painful cough, which the patient tries to suppress, when the breathing is impeded; hoarse grunting; diminished or lost appetite; difficult deglutition; retardation of defecation and urination; hard condition of the faeces; and yellow-brown (fiery) colour of the urine.

These symptoms naturally increase in intensity when the disease assumes a more severe type. The mucous membrane becomes deeply congested and livid; respiration difficult and almost spasmodic; the patient sits on his hind quarters with his head stretched forward and his fore legs wide apart, in order to get air into the lungs through the open mouth, from which panting and whistling sounds issue; there is great anxiety and uneasiness; the eyes are protruded and wildly staring; the veins of the head are swollen; the snout, lips and intermaxillary space down to the throat assume a purple colour; and the patient begins to stagger, falls down, and, becoming convulsed, dies from suffocation after the disease has run a course of from twenty-four hours to three days. If the disease does not terminate fatally, recovery may take place in from five to ten days with gradual decrease of the symptoms, in which case hard painless swellings frequently remain in the intermaxillary space, the presence of which often more or less obstructs the entrance of air into the larynx and imparts a panting character to the breathing. According to Röhl, these swellings are the remains of small, empty, circumscribed abscesses, surrounded by infiltrated connective tissue.

Besides excessive inflammation in the pharynx and larynx, *post mortem* examination occasionally reveals croupy deposits on the larynx and upper part of the trachea, which characterize so-called membranous angina (Sesselmann). We also find signs of asphyxia (excessive oedema, etc., of the lungs; hyperemia and its results in the veins of the brain).

## NON-INFECTIVE DISEASES.

*Prognosis* depends on the causes and the possibility of removing them, on the degree, and particularly, on the extent of the affection, and on timely application of appropriate treatment. The more the larynx is implicated, and the more dyspnoea present, the more unfavourable will be the prognosis, because suffocation is the most frequent and rapid cause of death. The degree of fever has also to be taken into account.

As regards *treatment*, it is necessary, above all things, to provide an abode which is dry, moderately warm, and free from draughts; and food consisting of meal and bran mashes, green fodder, or sour milk. At the beginning of the disease it is well to give an emetic, which sometimes does not act, and can be repeated next day. For this purpose white hellebore (8 to 30 grains) is the most appropriate agent, and should, if practicable, be given in the food. Ipecacuanha (15 to 45 grains) may be used instead. When the animal will not eat, the emetic may be mixed with fat. Drenches should not be employed; for which reason tartar emetic is not very appropriate. For these cases we can recommend the rectal application or subcutaneous injection of veratrin (.3 to .5 grains in a drachm of spirit). Apomorphine has no effect on pigs. If there is much difficulty in swallowing, we should abstain entirely from giving medicine by the mouth, for so doing would be almost certain to cause pneumonia. Occasionally the veterinary surgeon is called in too late for treatment, in which case the animal should be slaughtered as quickly as practicable, unless circumstances (pregnancy, state of nutrition, breeding value, etc.) suggest the expediency of giving it a chance of living. Stimulating inunctions (Ungt. Cantharid., etc.) on the region of the larynx have sometimes good results. Priessnitz's bandages are worthy of recommendation. Local treatment of pharyngitis is not usually practicable with pigs. Lövy states that he has seen good results from painting the pharyngeal mucous membrane with Lugol's solution. Meyer recommends energetic massage of the throat with rags saturated in paraffin oil.

2. *Pharyngitis of dogs* is also a not uncommon affection, and is one in which relapses are numerous. According to the statistics of the Berlin canine hospital, the frequency of pharyngitis in dogs amounts to one-third per cent. of all cases of disease. The finer breeds, such as pugs, terriers and greyhounds are most susceptible to it. The disease begins with impairment of prehension, difficulty of swallowing, and salivation. A painful swelling of the sub-parotid lymph glands, which in very rare cases suppurate, quickly makes its appearance. On inspection of the pharynx we can generally see severe congestion and swelling of the mucous membrane of the pharynx, tonsils, soft palate, etc. The course is usually benign, although the disease generally lasts for a considerable time (14 days to 4 weeks). Sometimes deafness occurs, apparently in consequence of the spreading of the inflammation of the pharynx through the Eustachian tube to the middle ear. The treatment of pharyngitis with little or no fever, consists chiefly in the application round the throat of Priessnitz's bandages, which should be removed every three hours. The swollen glands may also be rubbed with camphor liniment, etc. A local treatment of the affected pharyngeal mucous membrane can be practised by painting it with astringent fluids, such as lime water, and solutions of nitrate of silver ( $\frac{1}{2}$  to 1 per cent.), tannin, alum (1 to 2 per cent.), etc.

## CHAPTER II.

### DISEASES OF THE OESOPHAGUS.

**Classification.**—The occurrence of diseases of the oesophagus are as rare as their diagnosis is difficult. Some pathological processes, as, for instance, those caused by foreign bodies, belong more to surgery than to medicine. We, therefore, propose to discuss under this heading only the following affections of the oesophagus :—

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|---|--|
| 1. Dilatations.<br>2. Constrictions.<br>3. Lacerations. | 4. Paralysis.<br>5. Spasms.<br>6. Inflammations. |
|---|--|

#### DILATATIONS OF THE OESOPHAGUS.

**Etiology.**—The dilatations of the oesophagus may be divided into diffuse expansions which involve the entire periphery of the oesophagus, and into circumscribed ones of a certain part of the wall (oesophageal diverticula). The ectasis usually occurs along with stenosis of the oesophagus, and in that case always above it. The ectasis develops in a manner similar to that of a dilation of a ventricle of the heart with stenosis of the valves. The muscles of the oesophagus become gradually relaxed, owing to over exertion in trying to press accumulated masses of food through the narrowed part of the oesophagus, the food accumulates above the constriction, dilates the walls and causes the muscular coat to become atrophied and pale. Ectasis without stenosis is rare, and seems to depend on local inflammatory changes of the walls, or on injuries, as in the case of sudden expansions of the mucous membrane through the split separated muscular coat. Perhaps the presence of Meischer's tubes (*psorospermie*)—often found in the oesophageal muscular apparatus of the horse—plays a certain part in bringing about these dilatations.

Diverticulae are divided according to their mode of development into diverticulae by pressure and diverticulae by traction. The former are produced by the pressure from within of a ball of food on a part of the wall which is more yielding than the remainder, as a result, perhaps, of previous injury to the wall. The latter are usually the consequence of a cicatrical contraction of the peri-oesophageal tissue, or of a neighbouring organ, as, for instance, glands, in a manner analogous to the formation of bronchiectasis in cases of interstitial pneumonia.

The dilatations are either defined, in which case they are usually spindle-shaped, or they implicate the entire oesophagus. Stockfleth once found the oesophagus in a horse dilated to a length of 54 inches, with a diameter in the middle of  $3\frac{1}{2}$  inches. Diverticulae may hold as much as a bucket, and may become adherent to neighbouring parts, as, for instance, to the trachea. Dilatations and diverticulae of the oesophagus are not very rare in the domestic animals, and in horses and cattle are generally met with in the cervical portion of the oesophagus. They are also found in sheep, goats and dogs.

According to Storch some horses have congenital diverticula, or at least a predisposition to contract them, the second pharyngeal cleft probably participating in this formation. He also considers that the imperfect internal pharyngeal fistulae are perhaps connected with the pouch-like diverticula of the oesophagus.

**Symptoms.**—As these dilatations become developed very slowly, it is difficult at first to recognise their presence. The earliest marked symptom is a capricious appetite, which is manifested by excessive hunger on some occasions, and by rapidly produced satiety on others. Gradual emaciation sets in, and the rate of feeding often becomes very slow. This condition may last for years without its cause being recognised. In most cases it is accompanied by vomiting, which appears sooner or later after feeding, and recurs frequently, periodically and with a regular duration. Oesophageal regurgitation is sometimes effected through the nose, and is occasionally accompanied by a painful cough. The thrown-up masses are usually covered with frothy mucus, and if they have remained some time in the oesophagus, may be more or less decomposed; but they will in no case be digested, as has often been erroneously stated. This mistake has probably arisen from the fact that food may be brought up from the stomach at the same time. Independently of these

## DILATATIONS OF THE OESOPHAGUS.

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discharges of food, there is frequently a flow of much mucus mixed with food from the mouth and nose. Usually deglutition is very difficult or impossible. The animal sometimes tries with the greatest exertion to swallow a mouthful, while keeping his head and neck considerably flexed, and the hind legs placed under the body, in which case we often hear peculiar gurgling sounds. It sometimes happens that the animal, without having eaten, goes through the movements of mastication and deglutition, and at the same time slavers a good deal, and has frequent colicky pains, which often come on periodically. In cows tympanites, suppressed rumination, and, as some state, pica (p. 40) have been seen. In some cases the animal can readily swallow oats and chop, but not hay.

One of the most important symptoms of dilatation of the cervical portion of the oesophagus is the appearance, on the left side of the throat immediately after eating, of a swelling which can be emptied by pressure, and which may attain the size of a man's head, and may also be immovable. We may also find on the left side of the throat a longish tumefaction of the oesophagus, which, when oesophagitis supervenes, becomes painful to the touch, and exhibits very marked peristaltic movements during deglutition. On the introduction of a probang, we find an obstacle at the site of the stenosis, which is situated below the dilatation, or at the pouch-like diverticulum, or where the masses of food have accumulated. In rare cases the swelling occurs on the right side of the throat.

These symptoms of the digestive apparatus are very often complicated with cough, dyspnoea and attacks of suffocation, and even fatal asphyxia may arise from compression of the trachea by the enlarged oesophagus. In other cases fatal pneumonia, due to defects of swallowing, may also occur. In one instance strong pulsations were found in the left carotid artery. The disease, which usually lasts for years, produces gradual debility and emaciation, and may end fatally by suffocation, by pneumonia due to foreign bodies, or by rupture of the greatly distended oesophagus. Septicæmia sometimes appears to become developed from ulcerous changes in the oesophageal mucous membrane.

**Prognosis.**—Prognosis is nearly always unfavourable. This disease may easily be mistaken for rupture, inflammation, spasm, or paralysis of the oesophagus; abscesses in parts adjoining the oesophagus; pharyngitis; stomatitis; acute

gastric catarrh; poisoning and colic. The same remark applies to the other affections of the oesophagus. We can, however, make without difficulty a correct diagnosis by a careful examination of the oesophagus with a probang, and particularly by paying attention to the long continuance of the disease.

**Therapeutics.**—As treatment is not very successful, we have to rely chiefly on a fluid or semi-fluid diet, and on frequent feeding on small quantities at a time; and we may try, by means of the probang and by massage, to prevent occlusion by food. Little is known about the operative treatment of a diverticulum. Stockfleth, following the example of Moisant and Colin, recommends extirpation of the dilated part of the wall, and union, by suture, of the edges of the wound. Schindelka cured a case in a horse by these means. Tracheotomy instruments should be kept in readiness, on account of severe dyspncea being likely to supervene.

#### CONSTRICtIONS OF THE OESOPHAGUS.

**Etiology.**—Stenosis of the oesophagus may occur in various ways. The congenital form is the rarest. It may be caused by compression of the oesophagus from without, as in the case of tumours, such as strumata, actinomycomata, lymphomata, peri-oesophageal abscesses, especially in cattle; abscesses and tubercular degeneration in the bronchial and mediastinal glands; aneurisms of the aorta and melanomata. These tumours may exert pressure either laterally or in the form of a ring. Stenosis due to narrowing of the lumen of the oesophagus from within (obturation-stenosis) is usually caused by impacted foreign bodies (bones, potatoes, pieces of carrot, pieces of bread, cartilage, apples, physic-balls, pieces of food, broken-off tracheotomy tubes, etc.). It may also be produced by tumours which project into the lumen, as, for instance, palpillomata, carcinomata and actinomycomata in cattle; by tumours which contain spiroptera sanguinolenta, and by inflammatory hypertrophy of the muscles of the oesophagus in dogs. Also strictures may be set up by cicatricial contraction of the mucous membrane in consequence of erosions or injuries, or by a cancerous degeneration of the walls. They may take the form of a thick muff with a rough surface, or of a ring; or the thickened cicatricial tissue may project into the lumen in the shape of a valve. We shall discuss later on (p. 36) the stenoses

## LACERATIONS OR PERFORATIONS OF OESOPHAGUS. 33

which are caused by contraction of the oesophageal muscles. Particulars about foreign bodies in the oesophagus will be found in text-books on surgery.

**Symptoms.**—As the above-mentioned forms of stenosis are frequently complicated, later on, with dilatation, the clinical aspect of stenosis is, on the whole, not much different from that of oesophageal ectasis, the symptoms of which may be referred to here. Chronic tympanites is frequently a very characteristic symptom of oesophageal stenosis in cattle. As stenosis of the oesophagus in cattle is usually caused by the compression of a tuberculous bronchial or mediastinal gland, we can diagnose this form when other and less well-marked symptoms of tuberculosis (cough, emaciation, dulness on percussion, friction sounds, etc.) are accompanied by chronic tympanites, without any other digestive disturbances being present. (See chapter on tuberculosis, Vol. I. p. 152.)

**Therapeutics.**—The treatment of stenosis, and especially that of cattle, produced by compression and accompanied by tympanites, belongs to the province of surgery, and consists in the frequent use of the probang. We might, in a manner similar to the treatment of stenosis in man, try to widen the constricted part by the introduction of probangs of successively increasing thickness. The dietetic principles are the same as in dilatation of the oesophagus. When foreign bodies are in the oesophagus, we may try arecoline or pilocarpine.

## LACERATIONS OR PERFORATIONS OF THE OESOPHAGUS.

**Etiology and Symptoms.**—Tearing of the oesophagus from without inwards arises from external injuries, especially kicks by horses and falls. Perforations from within outwards may be due to impacted foreign bodies, careless introduction of a probang, and bursting of the very thin wall of a diverticulum, or muscular wall which has suffered from fatty degeneration, even if deglutition be normal. The symptoms vary according as the oesophagus is torn in its cervical part or in its thoracic part.

Rupture of the cervical portion of the oesophagus manifests itself, first of all, by a swelling which is often enormous, and which is caused by the escape of fodder, and is complicated later on with a phlegmonous inflammation. Swallowing being im-

possible, regurgitation of food and drink takes place through the mouth and nose. In some cases the entrance of air into the connective tissue which surrounds the oesophagus causes a diffuse emphysematous swelling of the throat, head and shoulders. The formation of an oesophageal fistula, and even of an oesophago-tracheal fistula has been observed, and in these cases the expired air has a putrid, gangrenous smell.

The cases of rupture of the thoracic portion of the oesophagus are still more severe. The animal exhibits sudden vertigo, and falls down during work, or presents to the spectator symptoms of violent congestion of the lungs and dyspnoea, and sometimes apparently those of colic. Pneumo-thorax is an occasional complication. In these cases the patient shows great anxiety, trembles, evinces a desire to vomit, and stands with the head stretched straight out, while the muscles of the neck are violently contracted. Symptoms of purulent ichorous pleuritis gradually appear, with the result that the animal dies.

**Therapeutics.**—Ruptures of the thoracic part of the oesophagus are not amenable to treatment; but an animal suffering from a rent of the cervical portion of the gullet may sometimes be saved by prompt operative treatment, which should consist of free incisions into the tumour and reuniting the adjusted edges of the wound by sutures. (See Sander's case in *The Veterinarian*, 1851.)

#### PARALYSIS OF THE OESOPHAGUS OR PHARYNX (*Dysphagia paralytica.*)

**Etiology.**—Paralysis of the oesophagus and pharynx often occur together, and consequently cannot be distinguished from each other in practice. The causes are to be found either centrally, in the brain, or peripherally, in local changes of the oesophagus and pharynx. The central causes are paralysis of the centre of deglutition in the brain in consequence of haemorrhage, concussion of the brain, abscesses, new growths, parasites, and particularly inflammation of the brain (subacute inflammation of the brain in horses). Paralysis of the centre of deglutition is observed in the course of some poisonings, such as pathological fungi (*tilletia caries*, and mould fungi), in herbivora, and ptomaine poisoning (sausage poisoning) in carnivora. Uterine paralysis and rabies are infective diseases which set up cerebral paralysis of deglutition. The local causes

consist in inflammation of the pharynx and oesophagus; due to the inflammation spreading from the mucous to the muscular coats. These inflammations are either primary or secondary, as when they occur during the course of strangles or petechial fever.

**Symptoms.**—The principal symptom of paralysis of the oesophagus and pharynx is inability to swallow, in consequence of which saliva and masticated food dribbles continuously from the mouth. In cases of the oesophagus alone being paralysed, it is sometimes filled with food from the pharynx down to the stomach, which condition is manifested externally by a cylindrical swelling on the left side of the throat. In other respects the symptoms are similar to those of other affections of the oesophagus. Occasionally fatal pneumonia, due to foreign bodies, appears later on.

Paralysis of the organs of deglutition is generally complete or bilateral; but in exceptional cases we may find a unilateral paralysis of deglutition, when the originating paralysis is restricted to one side of the pharynx. Schöttler describes a case of periodic oesophageal paralysis in a mare.

**Therapeutics.**—The treatment of paralysis of deglutition is confined within narrow limits. Besides the application of external irritants, such as blistering ointments, to the region of the pharynx and oesophagus, and the introduction of the probang, we may try internal stimulants in the form of subcutaneous injections, as, for instance, strychnine and veratrine. Electricity may also be employed. Sometimes a spontaneous recovery takes place.

We must not confuse inability to drink water with paralysis of deglutition, because otherwise healthy animals, which are able to masticate and swallow dry food in a normal manner, cannot suck up water, the functional failure being generally due to congenital causes, owing probably to some defect in the organ of suction. The affection appears not only in sucklings, but also in full grown animals, particularly horses. Straub has described such a case, and we have treated one in an eight-year-old horse, which was brought to us with the report that "it drank like a dog." This animal which was otherwise quite healthy and able to eat dry food in an ordinary manner, could not perform sucking movements. Although it played with the water and tried to drink, it was

quite incapable of doing so. The only treatment was to give the animal as much water as possible with its food.

#### SPASM OF THE OESOPHAGUS OR PHARYNX (*Oesophagismus*).

Independent spasmotic stenoses (*dysphagia spastica*), as compared to symptomatic spasmotic contractions of the oesophagus, with which they are often confused, occur but rarely. In most cases, a morsel of food which has stuck fast in the oesophagus, an injury to the gullet, or an ulcer, produces spasmotic conditions, similar to those caused by pure neuroses (*oesophagismus*), which Mollereau has observed in a very nervous Percheron; Guilmont, in a foal; and Mossé, in a hinny and in foal mule. The spasmotic attacks in these cases lasted from three-quarters of an hour to six hours. In the case of the Percheron, the animal was convulsed, vomited, placed its feet under its body, and stretched out its head. In the Guilmont case, the foal suddenly began to paw the ground and showed other signs of colic, could not swallow, and made attempts to vomit when one pressed on the tense oesophagus. The treatment consisted in the administration of bromide of potassium, chloroform, morphia, etc. Mossé effected a cure by oesophagotomy, and by the insertion of an oesophageal tube. Friedberger has observed an instance of periodic spasmotic stenosis in a horse. Cadéac has described a very interesting case of chronic spasm of the oesophagus in a well-bred horse, which, during a year and a half, had several attacks each week. While feeding, he would show great uneasiness, paw the ground with the fore-legs, stretch out the head, discharge frothy saliva from the mouth, and, when trying to vomit, utter shrill or sharp hissing sounds, while at the same time, saliva was ejected through the nose. During the attack, which lasted from one to two hours, the bolus of food could be seen moving violently up and down the oesophagus, and the upper lip was spirally contracted. The vomited food was on no occasion sour. The consumption of dry, short fodder and of cold water was shown to be the exciting causes of these seizures, for they were experimentally produced with certainty by giving such aliment.

Spasms of the swallowing muscles occur in rabies, epilepsy, catalepsy and tetanus, and require a casual rather than a systematic treatment. They arise chiefly from contraction of the muscles of the oesophagus, and perhaps of the muscles

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of the upper part of the gullet. The cases described as "intermittent paralysis of the oesophagus" are no doubt periodic oesophagismus.

### INFLAMMATION OF THE OESOPHAGUS (*Oesophagitis*).

**Varieties.**—Inflammation of the gullet may involve the mucous membrane, submucosa, muscles and periesophageal connective tissue, so that, strictly speaking, we have to recognise a catarrhal, phlegmonous and parenchymatous oesophagitis, and a peri-oesophagitis. The three last-named varieties are usually due only to foreign bodies or wounds.

**Causes.**—There are various causes of inflammation of the mucous membrane of the gullet. Thus, we find catarrhal oesophagitis occasionally in cases of pharyngitis, and also as a symptom of general diseases, such as rinderpest, foot and mouth disease, and sheep-pox. It may be caused by new growths in the oesophagus, or by parasites, especially by *spiroptera sanguinolenta* in dogs. In other cases foreign bodies, and especially corrosive substances, such as mineral acids and alkalis, set up catarrhal and even croupy inflammation of the mucous membrane. For instance, croupy inflammation has been observed in cattle after drenching with ammonia for tympanites; and ulcerous changes have been seen in horses after the administration of undissolved tartar emetic. Thermal irritants may also excite inflammation.

**Anatomy.**—The anatomical changes vary according to the intensity of the disease. In a catarrhal and somewhat superficial inflammation, we may find no increase of mucus, as in other inflammations of the mucous membrane, but only increased hyperæmia and considerable proliferation of epithelium. Chronic catarrhs are distinguished by their tendency to papillary proliferation. This peculiarity is explained by the fact that the oesophageal mucous membrane, especially in cattle, closely resembles skin in structure, a fact which Schütz has demonstrated by careful study. It possesses an epidermis, a rete malpighii, and papillæ supported by a layer of connective tissue. Therefore its pathological changes resemble those of the skin, especially as regards desquamation of epithelium and papillary hyperplasia in chronic catarrh. It is to be regretted that in most *post mortem* examinations too little attention is paid to the condition of the oesophagus, and for

this reason our knowledge of the anatomical changes in this organ are very incomplete. Kitt found in horses and dogs losses of substance in the form of "gnawed-out spots," a dull grey, greasy epithelial covering, congestion in streaks, petechial hemorrhages and swelling of the submucosa.

**Symptoms.**—The symptoms in mild catarrhs are not well marked. In the beginning of an attack it is hardly possible to make a correct diagnosis; but the appearance of difficulty of breathing, attempts to vomit, and increased sensibility of the gullet to pressure from outside, furnishes us with valuable diagnostic data. In a case of croupy inflammation of the oesophagus, which was caused by the corrosive action of ammonia, severe groaning, slavering, coughing and inability to swallow were observed. Also a croupy tube, 15 inches long, and having a thickness equal to the calibre of the oesophagus, was expectorated after a few days.

**Therapeutics.**—In those cases which can be diagnosed the treatment should be restricted to the application of cold in the form of small pieces of ice for the patient to swallow, non-irritating watery diet, and the internal administration of astringent solutions (tannin, alum, potassium chlorate, etc.), if they do not upset the digestion. Counter-irritation on the throat is useless. The periodic introduction of a probang may be used to prevent oesophageal stenosis, which might be caused by the cicatrisation of croupy ulcers.

#### DISEASES OF THE CROP OF BIRDS.

##### CATARRH AND INFLAMMATION OF THE MUCOUS MEMBRANE OF THE CROP.

**Etiology.**—Catarrh of the mucous membrane of the crop becomes developed by abnormal retention, drying, and decomposition of food in the crop, by the consumption of putrid food, by overloading, by accumulation of large masses of indigestible materials (such as sand and stones), and by the invasion of parasites, as, for instance, Spiroptera nasuta (*Filaria nasuta*), according to Robin, and *Filaria uncinata*, according to Zürn. Intense inflammation of the mucous membrane of the crop is found in cases of poisoning, as for instance, with phosphorus, arsenic, mercurial preparations, common salt, etc.

**Symptoms.**—In the not uncommon simple catarrh of the mucous membrane (so-called "soft crop"), we find a soft and sometimes a tympanitic swelling of the crop, on account of the consumption of food having almost entirely ceased. The bird tries more or less successfully to vomit, and eructates foetid gases, the discharge of which can be easily effected by pressure on the crop. The bird is very weak, and often dies from inability to take food.

**Therapeutics.**—Treatment consists of massage, with the object of pressing out of the mouth the contents of the swollen crop, while the head of the bird is kept hanging down. We may give with a teaspoon astringent solutions of alum, tannin, or sulphate of iron, or a solution of salicylic acid, and, after keeping the bird fasting for one day, we may, for the next few days, give soft food with only a few grains of corn.

#### OBSTRUCTION OF THE CROP (*Ingluvial Indigestion*).

**Etiology.**—The obstruction or "hardening" of the crop is usually caused by the consumption of substances which are more or less indigestible, such as hard, dry corn, and foreign bodies of an extremely varied nature, as, for instance, the leaves of *Robinia pseudo-acacia*. Pressure on the oesophagus, produced by inflammation in the adjoining structures, such as the lungs, pleuræ and trachea, may, in exceptional cases, cause overloading of the crop. Railliet and Lucet have described a case of ingluvial indigestion in ducks, which was set up by *trichosoma contortum*. These thread-like worms had settled under the mucous membrane of the crop, and caused the walls to become thin and hyperæmic.

**Symptoms.**—The most striking symptom is the extraordinary distension of the crop. Zurn saw a hen's crop which, with its contents (food, sand, small stones, etc.), weighed 2 lbs. 14 oz. We found on one occasion a hen's crop which had a circumference of 16 inches, and a diameter of 6 inches. In these cases, contrary to the condition of "soft crop" above, it is hard and firm to the touch, no food is eaten, and a discoloured, foetid fluid flows from the beak as a consequence of the catarrh which usually supervenes. Occasionally the tightly stretched wall of the crop becomes ruptured, or a so-called "pendulous crop" is developed, as the result of repeated dilatations of its wall.

**Therapeutics.**—The treatment, as in that of "soft crop," consists of massage in the form of rubbing, stroking and kneading. Some recommend the administration of hydrochloric acid, a 1 per cent. solution of which we generally use (a tea to a table spoonful every three hours). Zürn gives one or two drops of this acid in a teaspoonful of an infusion of peppermint or sweet flag (*Acorus calamus*), three or four times daily. As a last means, we may surgically open the crop and remove the accumulated matter. This operation, which should be carried out according to the principles of cesophagotomy, is not applicable to pigeons, because the results of many cases, with the exception of one reported by de Does, show that these birds bear it very badly. Zürn ascribes this susceptibility in pigeons to the peculiar condition of the membrane lining the culs-de-sac of the crop (accessory pouches), which, particularly during pairing-time, are very vascular.

#### VITIATED TASTE.

##### BOVINE PICA (*Licking Disease in Cattle*):

**General Remarks.**—Among the many pathological aberrations of taste in the domestic animals, the licking disease of cattle and the wool-eating of sheep occupy the first position, both from their scientific interest and from their importance in agriculture. Hence we have deemed it proper to deal with them in separate sections and, naturally, under the diseases of digestion. Other forms of morbid appetite, as in rabies, diabetes insipidus, etc., are merely symptoms, like the aberrations of taste which occasionally occur in mankind during pregnancy, chlorosis and hysteria, and do not demand separate discussion. The same remark applies to many other vitiated tastes which may be regarded as forms of play or vice, as, for instance, eating the after-births.

**Definition.**—Pica or licking disease is a peculiar chronic morbid condition which affects cattle more frequently than pigs, horses or goats, and manifests itself chiefly by an irresistible inclination to lick, gnaw and, if possible, eat all kinds of objects even of the most disgusting kind. It leads to disturbance of digestion and nutrition, to cachexia and finally to death.

**Etiology and Pathogenesis.**—Pica is more a manifestation of different morbid conditions, than a uniform independent disease, and may in this respect be compared to colic. Consequently, many different views have been taken of its nature. Thus it has been regarded as due to infection, heredity, imitation, nervous disturbance, brittleness of bone (*fragilitas ossium*), insanitary stables, and abnormal conditions of feeding, or of the soil upon which the food was grown.

1. *Infection and heredity* were formerly regarded as causes, without any valid reason, as exact observations have proved.

2. Although many have considered *imitation* to be a cause of pica, as it is of crib-biting, Lemke has proved experimentally that such is not the case.

3. Tscheulin and Spinola in particular, have looked upon pica as a nervous disease. Tscheulin considers pica to be a depraved condition of the sense of taste, and therefore discusses it in the chapter on nervous diseases. Spinola and others consider that it is due to an affection of the sensory nerves of the stomach and to a certain derangement of the pneumogastric nerves. Lemke has recently defined pica of cattle as "a chronic disturbance of nutrition produced by want of phosphorus, and set up by a primary disease of the nerves that control nutrition." It is certain that the nervous system becomes altered in this disease, and that the nerves of taste are irritated in a peculiar way; but it is extremely doubtful whether any case of pica is due to nervous disturbance. In accordance with the teaching of Haubner-Siedam-grotz'ky, it might be correct to say that some of the cases of pica are caused by peculiar conditions of nervous irritation.

4. Some of these must be referred to a disorder of the intestinal canal, especially when an animal under good hygienic conditions becomes affected, gradually grows thin, and finally dies of cachexia. If the administration of antidyspeptic and antidiarrhoeal remedies produce recovery in similar cases, we may feel certain that chronic dyspepsia or chronic intestinal catarrh was the cause of the disease.

5. Haubner and others regard bad stable conditions as a cause of the disease, which is a supposition that cannot be unreservedly accepted, because Lemke points out that it occurs in good stables just as often as in bad ones.

6. There is a great diversity of opinion with respect to the etiological connection of brittleness of bones with pica.

According to Roloff and Röll, pica is not an independent

disease, but merely the first symptom of fragilitas ossium, and may be the only symptom when the case terminates in recovery. Such instances have probably given rise to the idea that pica is an independent disease.

According to Rychner, Nessler, Anacker and Lemke, pica and fragilitas ossium are two entirely different diseases, each produced by a specific cause. Nessler points out that fractures, which are common in osteomalacia, are very rare in pica. Rychner and Anacker admit the occurrence of a complication of both diseases; and Lemke points out that pica becomes complicated with osteomalacia only when animals suffering from pica, are exposed to chill, and rheumatic influences.

According to Haubner-Siedamgrotzky, Spinola and others, pica may become complicated with osteomalacia, which will then form the termination of the complaint.

Our opinion on the question under discussion, is that in some cases, osteomalacia is the cause of pica. These two diseases are, however, separate morbid conditions, although they may be combined in certain cases. In practice we find pica without osteomalacia, and *vice versa*. Considering the close relationship between the two diseases, it is not improbable that the same cause might produce either.

7. The chief cause of pica is not to be sought in the food, nor in the soil which produced it; the defect being usually insufficiency of nutrient salts. We do not, however, possess exact information respecting the action of this cause. The researches of Nessler point particularly to deficiency of salts of sodium. At any rate, defective conditions of nutrition, which may be of different kinds, are the essential cause of pica.

In the first place, deficiency in the nutrient salts of the food produces an abnormal condition of the fluids of the body, which is a functional derangement that may be brought about by dyspepsia, as for instance, by intestinal catarrh, and probably by osteomalacia. In any case, the system will try to improve this general morbid condition. In addition to hunger and thirst, there will be a desire for certain nutrient salts. Although at first this desire is merely the result of an instinctive effort at self cure, it soon degenerates into vitiated taste, of which pica is a well-marked form.

Many of the statements that have been made respecting the connection between pica and food, are of a vague nature. It has by some authorities been attributed to feeding on

innutritious and indigestible food, such as hay which had hard stems, or which had been exposed to the action of rain, or was cut too late; straw; sour, boggy plants (*juncaceæ*, *equisetaceæ*, *cyperaceæ* and various kinds of *rumex*); sloppy and boiled food; sour food; too much or too little common salt; exclusive feeding on potatoes or roots, etc. Haubner went even so far as to attribute the development of the disease to certain plants, such as spicknel or hart's root, *meum athamanticum*, *achillea millefolium*, *Alchemillium vulgaris*, etc. This assumption is quite arbitrary, and has been refuted by the experiments of Lemke.

Nessler, who studied pica in the Black Forest, during three years, found by his analyses of hay and water consumed by the affected animals, a deficiency of salts of sodium, which he consequently regarded as the chief cause of the disease. The observations made by Nessler with respect to deficiency of phosphate of lime in food, refer to osteomalacia, the cause of which he also investigated.

**Occurrence.**—Bovine pica occurs sporadically as well as enzootically, and spreads widely in some years. It is a permanent resident in some districts, especially in the Black Forest and the Erz Mountains. As early as 1665-1721, Camerarius, in his *Ephem. Nat. Cur.* called the affected farms "*villa tubeficia*." According to Nessler, these farms are always on granite, and never on gneiss, which difference Nessler explains by the fact that gneiss is much more liable than granite to become decomposed by the action of the weather, and consequently it yields up its salts, particularly those of sodium, more readily to the soil and its plants. The disease often continues on these farms as long as 20 years, if the animals are not removed to another place. On other farms it appears only during dry seasons, when food is scarce. Haubner and Spinola state that in some places the presence of the disease makes it nearly impossible to rear calves, and even imported cows are attacked by it after two or three years' residence. Pica is specially prevalent in poor districts, on small farms, and among stall-fed animals, towards the end of the winter, when food becomes scarce. Cows in an advanced state of pregnancy, and those which give abundance of milk are more liable to contract the disease than others, probably because they need a larger supply of nutrient salts. Bullocks are not so often affected as cows. Young animals, especially

calves after weaning, suffer from pica, symptoms of which Lemke occasionally saw among sucklings.

**Symptoms.**—At the beginning of the attack, and for some time longer, there is no fever. Lemke divides the course of the disease into a first, or apyretic period, and into a second, or febrile period: The first perceptible disturbances consist in a somewhat diminished, or slower consumption of food, and in indolent and infrequent rumination. The appetite soon becomes capricious, and symptoms of pica make their appearance. Unless compelled by hunger, the animal will eat by preference large quantities of straw from the floor, and when pasturing will, contrary to its normal taste, prefer highly manured rank grass, and young sprouts of trees and shrubs, or in the stall try to approach the attendants in order to lick\* their clothes with peculiar eagerness. Day and night the animal continues to lick more and more persistently both bars, walls, woodwork, etc., frequently with loud clicking movements of the tongue, and will generally gnaw these objects as well, and even its iron neck-chain. At the same time there is increasing restlessness and excitement, and often abnormally great sensibility over the course of the vertebral column, especially in the lumbar and sacral regions. According to Lemke, these signs continue as long as four months, but vary somewhat in their intensity. At this period, as a rule, the state of nutrition is not much disturbed, nor the decrease of milk considerable, and the licking and gnawing can almost always be stopped by threatening the animal. The symptoms soon become aggravated, particularly the desire to gratify their depraved appetite, in consuming soiled litter, dung, decayed wood, old rags, ropes, leather, and especially earth, loam, sand, substances containing lime and clay, fragments of mortar and bricks, and any filthy water in preference to pure water. It even happens that affected cattle eat each other's hair. (Utz).

\* Even perfectly healthy cattle, especially if stall-fed, will often show extraordinary obtrusiveness and desire to lick the clothes of strangers going into the stall. These animals may also take up and swallow very strange things, without being in any way affected by pica. Markeische relates a strange case in point of a healthy cow which had never been ill, and which was one of the best feeders, devouring one after another, a pair of cotton stockings, cheese wrapped up in a cotton cloth, a waistcoat with metal buttons, a child's merino frock, and a woman's calico jacket, with the exception of the sleeves. Similar examples are found in Gurit's *Pathological Anatomy*.

In addition to decrease of appetite, in consequence of the constant licking and gnawing, which prevent the animal from obtaining the necessary time to feed, refusal of good food, and consumption of all kinds of indigestible and disgusting things, rumination becomes shorter and less frequent, and the movements of the rumen grow weaker and are sometimes temporarily suppressed. The milk becomes poor in fat (Lemke) although its quantity may continue normal for a long time (Leytze). The dung generally appears dry and may be even hard, and covered with considerable masses of ropy mucus, or semi-fluid and foetid. The urine is usually acid and sometimes contains albumen. At this period the temperature shows a rise of from  $1^{\circ}$  to  $3^{\circ}$  F.; and the rate of the pulse is from 70 to 80 a minute. The pulse is small and the arteries are contracted, but respiration does not seem to be much disturbed.

During the further course of the disease, the animal becomes quickly emaciated, almost to a skeleton, and especially after calving. The visible mucous membranes are pale, the hair lustreless, rough and erect; the skin, which is abundantly covered with dandruff, is dry and hard; and if we catch up a fold of the tense skin over the ribs, it will emit a crackling sound, and when we let it go, the fold will become effaced very slowly, showing that in this "hide-bound" condition the skin and subcutaneous tissues have lost a good deal of their elasticity. With increasing weakness and marasmus, the animal generally stands with its feet placed under its body and its back arched; it occasionally grinds its teeth, and its movements, especially those of the hind-quarters are stiff, narrowly limited in extent and accompanied by crackling sounds of the joints. Finally, the animal is unable to keep on its legs, it refuses nearly all food, the sounds of the rumen cease, and the animal dies from exhaustion.

**Course.**—The disease when left to itself is chronic, and its duration, according to Lemke, may extend from six months to two years. Spontaneous recovery may take place in from three weeks to four months (Lemke), especially in the early stages of the disease, by appropriate change of food or pasture, or changing from stall life to grazing in the spring, and *vice versa* in autumn. If the cause continues in action, and the animal be not properly treated, the case will generally end in death.

**Anatomy.**—In cases of slaughter, which are naturally those of an early stage of the disease, there is usually little to be seen, beyond slight emaciation and catarrhal changes in the mucous membrane of the alimentary canal. In animals which have died of the disease, we find extreme general emaciation; great deficiency of fat; a gelatinous condition of all the adipose tissue, which is frequently of a dun colour; the muscles greatly atrophied, flabby and pale; and the blood deficient in quantity, pale in colour, thin and clotting but feebly. We sometimes find slight transudations in the thoracic, abdominal and pericardial cavities, and a more or less severe catarrh of the mucous membrane of the gastro-intestinal canal. The cutis and subcutis, being in sympathy with the chronic disturbance of nutrition, are dry and hard. The changes which are said usually to occur in the bones, belong most probably to osteomalacia, and not to pica.

**Prognosis.**—There is no prospect of recovery, even in changing the food, if general emaciation and digestive disturbances are present. In slight cases a favourable prognosis depends on the possibility of removing the cause, especially by changing the food or pasture land. If the statements of Lemke with respect to the excellent results of apomorphine in the treatment of pica be confirmed, prognosis will be greatly improved, even when a change of food is impracticable.

**Treatment.**—It is first essential to discover the cause. Prophylaxis, as a rule, will be best carried out by improving the meadows and pastures by artificial manures, particularly nitrate of soda. We know from experience that the best and most natural means for combating the disease is to make a thorough change in the food, which we may effect either by obtaining food from districts that are free from pica, or by removing the affected animals to these places. As insuperable difficulties would usually attend the realisation of these measures, the efforts of the veterinary surgeon in ameliorating the faulty conditions of food are generally restricted to the addition of nitrogenous and leguminous foods, as well as mineral matters, more especially common salt.

From ancient times attempts have been made to improve digestion by the administration of weak hydrochloric acid,

small doses of common salt, bitters and aromatics, and in cases of excessive acidity, by antacids and absorbent means (lime water, chalk, magnesium carbonate, sodium bicarbonate, etc.), which treatment is particularly applicable to cases which have their origin in digestive affections. When osteomalacia is the cause, phosphate of lime is indicated. The administration of nauseous matters, such as garlic and stinking animal oil, has long been tried. Feser recommended the experimental use of hydrochlorate of apomorphine, which has been successfully used by Hackl and Reindl. In more recent times, Lemke has proclaimed it as the sovereign remedy for pica, and supports this statement by the results of a large number of experiments. Out of 226 cows, 21 oxen and 141 calves which Lemke treated by this means, all the animals without any exception recovered in a few days, and took only from eight to fourteen days to regain their appetite. For cattle which had been suffering for some time, he used apomorphine subcutaneously in doses of  $1\frac{1}{2}$  grains daily, on three consecutive days, and double that amount for those which had recently contracted the disease. Calves affected with pica require smaller doses. The curative effect of apomorphine remains in force only from three to five months, if the cause of the disease be not removed, but is otherwise permanent. Lemke also states that in districts where the cattle have usually to be changed every year or every two years, on account of the permanent prevalence of pica, they can be kept in comparatively good condition for at least three years by quarterly injections of this drug. The observations of Väth Gassner, Ostertag and Wagenheuser are very favourable to the efficacy of apomorphine in this disease; but Schuemacher, Röder, Flum and Hafner report failures or disagreeable results of this remedy.

**Pica in Horses.**—True pica is much rarer in horses than in cattle. Usually it is sporadic, and presents only slight symptoms. The cause is generally gastric disturbance, in which case the disease may be successfully treated by stomachics (neutral salts, Carlsbad salts, hydrochloric acid, etc.), or by antacids (lime water, carbonate and bicarbonate of soda, carbonate of magnesium, etc.). Steinmeyer obtained good results in a horse by the subcutaneous use of  $\frac{1}{2}$  grain of apomorphine in  $2\frac{1}{2}$  ozs. of water. The symptoms of the disease, which is found most frequently in foals, consist in persistent licking and gnawing of adjacent objects, or of the animal's own skin, dimi-

nution of appetite for normal food, and desire for eating litter and dung.

The eating of sand, which has been observed in many remount depôts, and which is the cause of "sand colic," may be attributed to play or vice in young horses left to themselves. It is found among those remounts that are left without supervision the entire day in paddocks laid down with sand, which they gradually learn to eat from mere *ennui*. The vice can therefore be checked in the majority of cases by stabling the animals and keeping them under strict supervision. The eating of sand is, however, sometimes a symptom of true pica, in which case it may be referred to deficiency of certain mineral substances in the food, and be treated by improving the soil, and by giving common salt, phosphate of lime, etc. We may also try to prevent the consumption of sand by sprinkling it with substances which have a disagreeable taste, such as tar, carbolic water, creolin water, wood vinegar, paraffin oil, etc., and by subcutaneous injections of apomorphine (three-fifths of a grain to three grains). An absolutely reliable remedy for sand-eating does not, however, exist. Foals have another bad habit of eating the hair of the tails of their mothers. This vice has nothing to do with pica.

**Pica in Pigs.**—According to Spinola, pica affects pigs in the same districts in which it attacks cattle, and is similar in both species as regards its course, duration and termination. It is stated that the animals eat by preference rotten wood and bark. Jansen states that symptoms of pica are seen in osteomalacia. Treatment consists in change of food, and in the administration of soluble bone meal. Emetics (tartar emetic, hellebore, ipecacuanha, veratrine, etc.) may be used experimentally.

The devouring of newly-born pigs by sows has as little connection with pica, as the eating of the after-birth by cattle. It does not seem to be the outcome of play or vice, but appears in the majority of cases to be due to temporary mental aberration, and occasionally to digestive disturbance, as constipation, for instance. Block recommends an emetic as a simple and reliable remedy. After removing the young ones, milk containing tartar emetic and powdered hellebore root (of each eight grains) may be administered to the sow, which by this means will become tranquil, and will receive her litter kindly. An aperient (calomel) and green food may also be prescribed.

WOOL-EATING OF SHEEP (*Ovine Malophagia*).

**Etiology.**—The eating and gnawing of wool by sheep is an affection closely allied to pica of cattle, and is of great economic interest, owing to the destruction and loss of wool entailed by it. Some say that it is a cause of death in lambs. Opinions on the cause and nature of wool-eating are as various as those regarding pica. The two principal theories, namely, those of imitation and mal-nutrition, though opposed to each other, may be both substantially correct, for their respective factors may co-operate in producing the disease.

1. The *imitation* theory has its chief partisan in Spinola, who assumes that *ennuis* prompts the first animal to eat wool. The following facts seem to indicate that the vice arises from imitation:—Firstly, only one sheep as a rule begins to eat wool. Secondly, Spinola was able to see the vice only in certain pens on the same sheep farm. Thirdly, the spreading of the disease in a herd was checked or entirely stopped by a strict separation of the wool-eaters (Spinola and Burmeister). Fourthly, the wool-eating occasionally begins even before the sheep are housed for the winter.

2. For many years the affection was ascribed to *deficient and faulty feeding*. This is a theory particularly advocated by Lemke, who believes that his experiments prove that it cannot be caused by imitation. It is especially frequent during winter, when fodder is insufficient in quantity, and largely supplemented by potatoes, potato "mash," and other food poor in nitrogen; but it disappears with a change of food. In lambs it is ascribed to deficiency of milk, starvation of the ewe, or to an abnormal condition of the ewe's milk. May states that on a sheep farm the lambs became wool-eaters on account of not getting enough milk from the ewes; but that they gave up the vice as soon as they experimentally received an addition of cow's milk. Haubner-Siedamgrotzky attributes it also to peaty, sour, wet pasture, and a deficiency of common salt.

**Occurrence.**—Wool-eating is found principally in finer, and especially in the merino breeds, and, according to the unanimous statements of all authors, only as long as the sheep are stall-fed during winter, namely, from autumn to spring, but rarely while they are at pasture. The affection frequently

appears within eight to fourteen days after the sheep have been housed, but disappears just as quickly when they are turned out to grass. Even putting them on corn during the winter has an immediate good effect.

**Symptoms.**—In cases of wool-eating by lambs, the animals are said to begin at the age of from two to six weeks, by gnawing the wool of their mothers, preferably that of the legs, belly and tail. At first only a little wool is gnawed off in play; but the desire gradually increases, and the lambs finally gnaw other sheep. On the whole, they appear healthy, without any marked disturbance of appetite or growth. These ill-results are evident only when the lambs, as sometimes happens, consume a great deal of wool, especially if it be long and coarse. They then become emaciated, are backward in development, constipated at times, and may even die with symptoms of gastro-intestinal inflammation. On *post mortem* examination, besides signs of inflammation, especially of the abomasum, we find pellets of wool varying in size from a hazel-nut to a walnut, and quantities of other wool, which are supposed sometimes to get into the pylorus and obstruct it.

If wool-eating appears in a flock of yearlings and older sheep, only one animal as a rule begins to eat the wool of the others, apparently as if seeking for forage in the fleece. It is stated that wool soiled by urine and dung is preferred. The first wool-eater often gnaws several sheep, but finally confines its attention to one. Others, one by one, soon join in and help him in working on the selected sheep. As a rule, they do not begin on another until they have cropped the first one bare. With the increase in the quantity of wool-eaters, the number of those selected to be clipped becomes greater and greater, with a corresponding loss of wool. As a rule, the wool-eaters remain apparently healthy, free from fever and loss of appetite, and their rumination is not disturbed. Occasionally some of them suffer from constipation, dryness of the wool, etc.; although wool-eating does not lead to well-marked cachexia and marasmus, ending in death, as in pica of cattle, and it does not set up osteomalacia or osteoporosis.

If the vice be but slightly developed, it is practised only by day, and generally in the intervals of feeding; but if it be in a severe form, it may be carried on also by night and in dark stalls (Lemke). Cases in which sheep nibble their own wool are extremely rare; consequently, this affection is not liable to

be confounded with scab and certain eczemas, which are accompanied with pruritis.

**Prognosis.**—Although mallophagia is a much milder disorder than bovine pica, death may occur from it among lambs. The remarks made on pica with respect to change of stalls, food and pasture, and the use of apomorphine, also apply to the prognosis of wool-eating.

**Treatment.**—Change of food and a liberal supply of nutriment are the first consideration, especially with lambs, and grazing is preferable to stall-feeding, if the weather permits: May recommends that the lambs be separated from the ewes after the third week from birth, but should be allowed to suck several times during the day.

No time should be lost in separating the first wool-eaters and the sheep that have been cropped from the remainder of the flock. The administration of common salt, alkalies, stomachics, etc., may also be tried. Spinola recommends that the stalls be kept dark during the intervals of feeding in the day, which is a measure not likely to be of much use in advanced cases.

Lemke recently obtained just as good results in cases of ovine mallophagia, as he did in those of bovine pica, by the subcutaneous use of hydrochlorate of apomorphine; in fact, he cured 800 wool-eaters by this remedy, without a single failure. He uses the same doses (1-5 to 3 grains) for sheep as for cattle. The curative effect of this drug is said to last for four months.

**The Plucking out of their own feathers by Birds.**—This vice is produced by *ennui* and desire to play, and is generally incurable. It is practised chiefly by parrots and canaries, and sometimes by cocks and hens and other poultry. We have unsuccessfully tried external applications of the tinctures of veratrin, asafoetida, aloes and gentian and other bad tasting fluids. We believe we have obtained cures in certain cases by the internal administration of apomorphine ( $\frac{1}{4}$  grain in  $3\frac{1}{2}$  oz. of water). Treatment is favourably influenced by change of food, (the principal food for parrots being boiled maize, hemp seed, and wheaten bread). This plucking of feathers must not be confounded with moulting during the course of certain skin affections.

## CHAPTER III.

### ACUTE CATARRH OF THE STOMACH AND INTESTINES IN HORSES.

(*Gastro-enteritis catarrhalis acuta—acute Dyspepsia.*)

**Preliminary Remarks.**—In this chapter we intend to deal collectively with acute catarrh of the stomach, acute intestinal catarrh, and the various abnormal conditions of the stomach which are known as indigestion, impaction of the stomach, dyspepsia, weak digestion, difficult digestion, gastricism, gasteritis, status gastricus, gastric fever, etc. In this respect we follow the old routine, which is based on the fact that it is extremely difficult in the horse accurately to locate the site and determine the intensity of a disease of the stomach or intestines. There is no doubt that in our domestic animals an acute catarrh may be confined either to the stomach or intestines, or may be restricted to certain parts of the intestines. Also, there are many stages between the physiological hyperemia of the mucous membrane of the stomach during digestion, and intense inflammation of that membrane. As vomiting, which is a characteristic symptom of gastric catarrh in other animals, is almost always absent in horses, and as the diarrhoea of intestinal catarrh is often not apparent in them, owing to the great length of their intestines, it is hardly possible in horses to distinguish between gastric catarrh and intestinal catarrh, which diseases very often succeed each other, or are simultaneously produced by one and the same cause.

We are also in want of reliable diagnostic means to distinguish between individual degrees of intensity. Formerly the mildest form of gastric disease was called dyspepsia, which was held to mean only a disturbance of the physiological function of the stomach, without any demonstrable changes in the structure of the mucous membrane. In the domestic animals the presence of such a form of dyspepsia may be suspected, but cannot be proved, although in all probability it occurs in old and anemic animals during convalescence after a long-standing disease. It is quite impossible to say where dyspepsia ends

and catarrh begins, especially as the former, if it be present for a long time, may change into the latter. Therefore, we have not dealt with dyspepsia as an independent disease. On the other hand, it is comparatively easy clinically to distinguish between mild gastric catarrh and severe gastro-enteritis, which we have therefore separated from the former, and will discuss separately later on. The more intense inflammatory affections of the gastro-intestinal mucous membrane are characterised by high fever and grave general disturbance, and can be distinguished from simple catarrh, not only anatomically, but also clinically.

**Etiology.**—The causes of acute gastro-intestinal catarrh, which is a very common disease in domesticated animals, and especially among horses, are partly certain noxious matters that exercise a direct influence on the mucous membrane, and partly factors of a more indirect nature. Special susceptibility to the disease is found in some individual animals, such as weakly, anaemic, badly-fed, pampered foals, very old horses, convalescents from severe diseases, animals whose constitutions have become weakened by relaxing food, and those whose powers of resistance have been diminished by previous attacks of gastro-intestinal catarrh. Some individuals of the finer breeds and those which are accustomed only to the best food, are very liable to contract the disease from slight causes.

Among the direct irritants of the mucous membrane we may mention water, and fodder which is too hot or too cold; grass which is frozen or covered with hoar frost; forage which is indigestible, irritating, mouldy, decomposing, too rich in lignin, or which contains poisonous plants (*colchicum*, *tobacco*, *digitalis*, etc.); sour milk in the case of sucklings; improper use of acrid and purgative medicines; exclusive feeding on maize, without giving salt; consumption of too much corn, bean or peas; imperfect mastication of food, owing to teething, old age, or voraciousness, and errors in feeding. Cases of acute gastro-intestinal catarrh are sometimes seen after feeding on new hay.

As indirect influences we have over-exertion, and work given too soon after feeding, in which case digestion is disturbed by the blood being drawn away from the organs of digestion to the muscles of locomotion. Climatic and atmospheric influences may also predispose an animal to this disease by chill or by weakening the constitution.

Acute gastro-intestinal catarrh sometimes appears epidemically and enzootically. This so-called infective gastro-

intestinal catarrh is caused by the entrance into the alimentary canal, of micro-organisms about which we possess but little information. In its initial stage, it is difficult to distinguish this condition from the others.

Besides these cases of independent acute gastro-intestinal catarrh, we find a symptomatic form of this affection in the course of different febrile diseases, such as pleuro-pneumonia, distemper, puerperal fever, petechial fever, etc.

**Anatomy.**—As the primary acute catarrh rarely causes death, *post mortem* examinations, as a rule, are made only in cases of secondary catarrh. Even then we may have a negative result, because the inflammatory changes that are present during life sometimes disappear more or less after death.

I. *Appearances in the Stomach.* The true mucous membrane of the stomach (that of the pyloric half) shows signs of catarrh, inflammatory swelling and severe congestion, which is sometimes diffuse and at other times circumscribed and spotted.

These haemorrhages, which vary in size, are sometimes punctiform, lenticular, or even larger, and give the mucous membrane a dotted, sprinkled or spotted appearance. The mucus is increased in quantity, is sometimes glairy, at other times purulent; may be stained with blood in consequence of slight haemorrhages, and may contain cellular constituents, mucous corpuscles, leucocytes, and desquamated and decomposing epithelial cells. Microscopical examination of the affected mucous membrane reveals mucous degeneration of the columnar epithelium of the excretory ducts of the glands, considerable granulation of the cardiac glands, cellular infiltration of the inter-glandular connective tissue and submucosa, with swelling and desquamation of the endothelium of the lymph vessels.

*Appearances in the Intestines.* The pathological changes in the intestines are similar to those in the stomach. The mucous membrane is inflamed, swollen and loosened, in consequence of the serous infiltration of the submucosa; the muscular wall of the intestine contains an abnormally large amount of fluid, is consequently tender, and easily lacerated. The inflammatory hyperæmia is sometimes narrowly limited in extent; at other times it involves large portions of the intestine, but seldom invades the entire intestinal canal. Sometimes the inflammation is confined to the intestinal villi, which become enlarged and elongated, and project in a striking manner into the lumen of the intestine, or the congestion takes

the form of an area around the more or less swollen Peyer's patches. In consequence of the swelling, these follicles sometimes project above the mucous membrane in the form of light grey nodules, which vary in size from a grain of millet to a hemp seed, and are surrounded by a red area, in which case the name of "follicular catarrh" has been given by some authorities to the condition. If these inflamed follicles become destroyed by suppuration or by attrition, small crateriform ulcers will be developed on their respective sites. These losses of substance, which may be called follicular ulcers, must not be confounded with true intestinal ulcers. In severe cases we may find diffuse epithelial desquamations (catarrhal erosions). The affected mucous membrane excretes a fluid which is rich in epithelium, and which may be serous, mucous, or purulent, according to the quantity of its cellular constituents. When haemorrhages are present, its usual light grey or buff colour may become tinged with red. Sometimes the desquamation of epithelium is particularly abundant (desquamative catarrh).

**Symptoms of Acute Gastric Catarrh.**—The first and often the only symptom is irregularity of the appetite, which may be entirely suppressed or diminished, capricious and even abnormal, as we may see by the animal's preference for rough food, straw and soiled litter. Thirst is more or less suppressed. On account of irritation to the terminal branches of the pneumogastric nerve, the animal may yawn uninterruptedly for hours. In the horse, overloading of the stomach may give rise to eructations and actual vomiting, without any great danger to the patient, and without signs of severe colic. We learn from veterinary literature that such cases of uninjurious vomiting are by no means as rare as is generally supposed, particularly in mountainous districts where horses at grass sometimes consume a large quantity of the leaves of white hellebore.

As a rule, the mucous membrane of the mouth presents important changes, and is either highly congested or pale. It is sometimes of a dirty yellow or livid colour. At first it is often very dry, and later on may be covered with a greasy, pappy, lather of frothy mucus, which has an insipid sweetish smell, or we may find an accumulation of glairy mucus, alkaline in reaction. Also, a swelling of the submucous venous network on the hard palate is often observed. The tongue is frequently coated, on account of the diminished consumption of food. Acute gastric catarrh is very often complicated with catarrh of

the entire buccal membrane. At first the abdomen is not much affected, but later on it becomes empty and tucked up. Peristalsis may be normal, or may be decreased from the very beginning of the attack. The faeces are often balled, dry, covered with a shining crust, are passed in small quantities at a time, often at short intervals, and frequently contain undigested particles of food. Colic is absent, as a rule, and the urine is nearly, if not quite, normal.

The mental condition varies according to the severity of the affection and individual idiosyncrasy. The animal is dull, depressed, easily fatigued, sweats with the least exertion, and breathes with difficulty if put to work. In many cases there is no rise of temperature; it is seldom very high. Usually the temperature of the rectum is not increased more than one degree Fahrenheit, and the pulse not more than eight to ten beats. If fever appears, it generally lasts for only a few days. In very rare cases, rigors may appear as a preliminary symptom. On the other hand, we often find unequal distribution of temperature, as may be shown by coldness of the ears and limbs, and by congestion of the conjunctiva.

**Symptoms of Acute Intestinal Catarrh.**—Contrary to what is the case in acute gastric catarrh, the appetite may be normal in acute intestinal catarrh. It is, however, as a rule, more or less disturbed, especially in the later stages of the disease. There is frequently great thirst if diarrhoea be present. The general health may remain unchanged, without any appearance of fever, although the total constitutional disturbance is usually more severe than in catarrh of the stomach. The chief symptoms arise from the intestine. Peristalsis becomes more active; the intestines give out rumbling and gurgling sounds which are often audible at a distance; but at certain times the intestinal movements may be stopped, and no sounds given off. The abdomen is occasionally tympanitic, and, in that case, painful on palpation. The dung is at first in the form of large soft balls, frequently covered with glairy mucus, or enveloped, as in a net, in dirty white or intensely yellow membranous, croupy masses (*Proctitis*). It usually contains a lot of undigested food, and emits an acid or putrid smell. Occasionally, after passing faeces, the animal discharges a greenish-yellow fluid, which soils the parts adjoining the anus, tail and inner surface of the thighs. Flatus, which generally has a foetid

smell, is often passed during defecation, and also independently of it.

Later on we see diarrhoea, which is a very important symptom for the diagnosis of intestinal catarrh. The faeces, which at first were soft and moister than usual, assume the consistency of cow-dung, and finally become thin and quite fluid. They give off a remarkably disagreeable acid smell, and are sometimes passed involuntarily through the gaping anus, and under conditions of great weakness (colliquative diarrhoea). The reaction of the faeces is usually acid (from the presence of fatty acids), which is a fact of no importance in diagnosis, because they often have this reaction in normal conditions. In some cases there may be no diarrhoea. If the catarrh be confined to the small intestine, the excess of fluid in the chyme passed onwards by it may become entirely absorbed in the large bowels, and consequently the dung may be discharged in balls. We should bear in mind that the presence of diarrhoea does not always imply the existence of a catarrhal intestinal affection. Apart from the fact that the faeces may be very soft when a horse is fed on green food, temporary diarrhoea may appear in consequence of increased peristalsis from reflex action, or as a result of temporary hyperæmia, due, for instance, to chill.

The urine, which suffers little or no change during gastric catarrh, manifests various abnormal conditions in acute intestinal catarrh. If diarrhoea be present, it becomes diminished in quantity, with a corresponding rise in specific gravity, often changes in colour, loses its sediment and becomes clearer. The amount of indican contained in it is increased. The most noteworthy point about the urine is, that it is abnormally rich in phosphates, only traces of which are found in healthy urine. Its reaction, which in normal conditions is alkaline, is neutral or acid.

**Duration and Progress.**—The majority of cases of acute gastro-intestinal catarrh which are accompanied only by slight or moderate fever, recover quickly, even in a few days, and seldom continue beyond a week. The fever gradually subsides, the appetite improves, the evacuations diminish in frequency, and the faeces become more solid. Occasionally the diarrhoea is followed by slight constipation. The return of the urine to its normal alkaline condition indicates improvement. In severe cases the catarrh becomes chronic or increases in intensity; the diarrhoea permanent, dysenteric and fluid; more or

less severe and frequent colic supervenes; emaciation sets in, the flanks become tucked up; the coat rough and staring; and the temperature of the limbs, cold. These symptoms are combined with rapidly increasing weakness and depression. The animal stands dull and listless in its stall, and shows its debility and exhaustion by lying down, and by frequently lifting up and putting down its hind legs alternately, so as to avoid placing weight on them. Appetite is then entirely lost. If recovery takes place in such a case, convalescence will be greatly protracted. Death will be the usual result under such conditions, especially with debilitated and old horses.

**Differential Diagnosis.**—It is hardly possible to mistake gastro-intestinal catarrh for other affections of the intestines, if no fever be present. As it is often difficult to decide whether the gastro-intestinal catarrh is independent or whether it is a symptom of a graver disease, we can only diagnose the former with certainty when we are sure that the latter is absent. Difficulties may also arise in distinguishing a severe case of gastro-intestinal catarrh from inflammation of the stomach. Here the diagnosis will depend on the further course of the disease, the degree of the fever, the gravity of the general symptoms, and the consideration of their causes. If, during the course of an acute catarrh of the large intestine, follicular ulceration becomes developed, it will in some cases be impossible to distinguish the supervening dysenteric diarrhoea (which has also been called catarrhal dysentery) from true infective dysentery. Here also, the further course of the disease, the history of the case, etc. will be the data for diagnosis. There are also some important points with respect to the localisation of the catarrh on different parts of the intestinal canal:—

1. When jaundice appears, we may conclude that the disease is located in the small intestine; because jaundice is brought on by absorption of bile caused by the orifice of the biliary duct becoming closed through swelling of the inflamed mucous membrane. If jaundice be absent, we may infer the presence of catarrh in the small intestine by participation of the neighbouring stomach, and by the remarkably active peristaltic movements of the small intestine, which will be manifested by loud rumbling sounds that can be best heard in the region of the false ribs and flanks.

2. If the large intestine be the chief seat of the disease, severe colic and diarrhoea will appear. A very characteristic symptom,

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particularly of the posterior portion of the large intestine (rectum and its adjoining parts), is the fact of the faeces being covered by membranous or retiform slime, consisting of mucus and epithelium, which does not penetrate into the dung. In well-marked catarrh of the rectum, the faeces are passed with abnormal frequency and only in small quantities at a time, even in single balls, defecation being accompanied by violent straining, groaning and arching of the back.

**Treatment.**—The prophylaxis of acute gastro-intestinal catarrh consists in attention to hygiene. Treatment in the first place should also be of a dietetic nature. In many cases such treatment suffices. We should give small rations of easily digestible food and, especially in cases brought on by overfeeding, should allow the horse to fast for some time before giving food. We should give only "chilled" water; should keep the animal comfortably clothed and have him wisped or hand-rubbed all over several times a day. Another important point is to take the patient for a short walk in the open once or twice every day. The medicinal treatment depends on the indications.

1. In impaction of the stomach the first thing to do is to remove the excess of food as quickly as possible, which we may by a dose of tartar emetic  $1\frac{1}{2}$  dr. and sulphate of soda 10 oz., or by giving 45 grains of calomel. Care has to be used in the administration of physostigmine ( $1\frac{1}{2}$  gr.) and arecoline ( $1\frac{1}{2}$  gr.), as they may cause rupture of the stomach, when that organ is greatly overloaded and distended.

2. In cases of abnormal fermentation in the stomach and intestine, we strongly recommend dilute hydrochloric acid (2-4 drams in the drinking water as a dose). Common salt (4 dr. to 1 oz.) and bicarbonate of soda ( $\frac{1}{2}$  to 1 oz.) also render good service. In cases of gastro-intestinal catarrh with flatulence and acidity of the intestinal canal, we have had good results with hyposulphite of soda. Creolin, which is a good antizymotic, can be given in doses of 3 to 6 drams, (creolin 2 oz.; glycyrr. rad. 4 oz.; Extr. glycyrr. et ceræ flavae q.s. to be made into 6 balls, 1 or 2 to be given daily).

3. If dyspepsia prevaii, we may try (besides hydrochloric acid) true stomachics, such as pepsin (1 to 2 dr.), common salt, Glauber's salts, sal-ammoniac (sulphate of soda 10 oz. chloride of sodium, 4 oz.; chloride of ammonium, 2 oz.; a tablespoonful at each meal), mustard ( $\frac{1}{2}$ -1 oz. powdered and given

as an electuary), pepper, ginger, tincture of veratrum ( $\frac{1}{2}$ -1 oz.), acorus calamus ( $\frac{1}{2}$  oz.), rhubarb (pulv. rad. rhei. 225 gr.; sodii bicarb. 150 gr.; pulv. rad. glycyrr. 150 gr.; pulv. rad. althraæ et aqu. font. q. s. f. electuar. to be given in 3 doses), aloes ( $1\frac{1}{2}$  dr. in a ball), gentian and aniseed.

4. In cases of diarrhoea, when only small quantities of foetid faeces are passed, we recommend at first, for the clearing out of the intestinal canal, small doses of calomel (30 to 75 gr.), which can be given even when follicular ulcers are present. We should then try to stop the diarrhoea by dietetic means, such as dry food, parched flour and parched oats. If we do not attain our end by these means, we may resort to astringents, such as oak bark, cinchona bark ( $1\frac{1}{2}$  oz.), sulphate of iron (2 dr.), or alum ( $\frac{1}{2}$  oz.), tannin (3-4 dr.), sugar of lead (75 gr.), and nitrate of silver (7 to 15 gr. in a ball, or dissolved in a large amount of water. Opium is certainly the best curative agent in severe diarrhoea, and may be given in a ball (75 to 300 gr.), or as tincture of opium (1 to 3 oz. as a single dose) in a mucilaginous decoction. We have given as much as 10 dr. ourselves without any unfavourable consequence. Trasböt advises an emulsion of 150 gr. of camphor, 150 gr. of asafetida, the yolks of 2 or 3 eggs, and a quart of rice water, to be given twice daily. Röll strongly recommends liquor strychnæ.

5. With cases of catarrh of the large intestine involving the rectum, we should supply local treatment by means of enemas. Even with ordinary diarrhoea, enemas of nitrate of silver are often much better than giving this drug by the mouth. Enemas are particularly beneficial in chronic cases of intestinal catarrh. In this manner we may employ 1 to 2 per cent. solutions of alum or tannin in catarrhal proctitis.

#### CHRONIC CATARRH OF THE STOMACH AND INTESTINES IN HORSES.

(*Gastro-enteritis catarrhalis chronica, or chronic dyspepsia.*)

**General Remarks.**—We have already explained that it is impossible to make an exact differentiation between chronic gastric catarrh and chronic intestinal catarrh, both of which taken conjointly may be termed chronic dyspepsia (indigestion), which is a common disease among horses. It is probable that some chronic catarrhs of the stomach and intestines are merely symptoms of other diseases, such as organic diseases of the stomach and intestines, and affections of the lungs, heart and kidneys. This is a diagnostic fact that deserves special attention in the case of old horses.

**Etiology.**—Some animals are specially susceptible to chronic catarrh. Continued mal-nutrition, anæmic and cachetic conditions such as those produced by severe illness, chronic poisoning, over exertion, etc. predispose an animal to chronic gastro-intestinal catarrh. Chronic catarrh often arises also from the acute form, when the dietetic causes which have been discussed under the heading of acute catarrh are continued or often repeated. The horses of millers frequently get this disease from almost exclusive feeding on bran and short-cut straw. Chronic catarrh of the stomach also arises from persistent wind-sucking.

Dental defects, especially in old horses, often cause irritation of the intestines, on account of the food being imperfectly masticated and insufficiently mixed with saliva.

Also, chronic catarrh is gradually developed by conditions which cause a permanent congestion of the mucous membrane of the stomach and intestines, such as frequent previous attacks of acute catarrh; congestion of the portal system, caused by chronic diseases of the liver, and more rarely by disease of the portal vein, congestion of the posterior vena cava in consequence of affections of the heart or lungs (valvular defects, pulmonary emphysema, and induration of the lungs), congestion of the intestine itself, especially circumscribed hyperæmia of the intestine by embolic obstruction of certain arteries in verminous aneurism. In all these cases the catarrh is not the immediate consequence of the congestion; but is rather a secondary affection, in that the intestinal mucous membrane suffers nutritive disturbances in consequence of the abnormal distribution of blood, and thus becomes less resistant to external irritants. Therefore, catarrh is often absent in such cases of venous congestion in the intestine. In this way chronic diarrhoea, brought on merely by frequent stimulation of the peristaltic movements of the intestines (as for instance, by chill), and the congestion consequent thereupon, occasionally set up chronic catarrh.

Sometimes chronic catarrh is due to the presence in the stomach and intestines of injurious entozoa, especially bots, which as a rule, however, are not hurtful, but sometimes, particularly when very numerous, produce considerable digestive disturbance, especially in foals. Besides *Gastrophilus equi* and *pecorum*, we have also to consider the larvae of *Gastrophilus haemorrhoidalis* and *nasalis* which are often found in great numbers in the small intestine. Chronic gastric catarrh is

occasionally set up by *Filaria s. spiroptera megastoma*, which is usually met with in the right sac of the stomach in the form of submucous cysts, varying in size from a bean to an apple. Respecting true entozoa (*Ascaris megalocephala*, *taenia mamillana*, *plicata* and *perfoliata*) see the chapter on helminthiasis.

Symptomatic chronic gastro-intestinal catarrh is caused by various organic changes in the stomach and intestines; as for instance by ulcers, dilatations, stenoses, displacements, new-growths, foreign bodies (concretions and calculi), impaction of food, affections of the pancreas (e. g. colloid carcinoma) and new-growths of the liver. Chronic catarrh of the stomach is found as a symptom of various chronic diseases, such as anaemia, rickets, and leucæmia.

*Kumgata disease*.—Under this name ("sand-disease") Vedernikoff has described a fatal disease which occurs among the horses of the Kirgis steppes, and which is accompanied by chronic gastric catarrh. On *post mortem* examination, large quantities (up to 10 to 12 lb.) of sand mixed with food are found in the stomach along with inflammatory changes of the mucous membrane.

**Anatomy.**—I. *Condition of the stomach.* The anatomical appearances vary according to the severity and duration of the complaint. In mild cases nothing important is to be seen. The changes are usually found in the mucous membrane of the right sac, and especially on the great curve, and in the neighbourhood of the pylorus. The mucous membrane is covered with a layer of ropy, glairy mucus, which may be of a light grey colour on account of the abundance of its contained epithelium, and which adheres firmly to it. The colour of the mucous membrane is by no means a reliable sign, and we must avoid mistaking normal irregular colouring of the right sac for pathological alterations. In chronic catarrh, the mucous membrane usually varies in colour from a dull red to a dirty red brown, and is sometimes slate-grey and spotted, so that it assumes a marbled appearance. The pigmentation is due to the deposition of black pigment granules, which come from the haemoglobin of extravasated red blood corpuscles, or from capillary haemorrhages. In other cases we find the mucous membrane to be of a pale buff colour, and intersected by a few greatly dilated blood vessels. The surface of the mucous membrane is either even and smooth, or peculiarly uneven and rugged, corrugated, papillary and

warty, or it forms stiff folds and ridges, and in rare cases, cone-like proliferations (*gastritis polyposa—polyposis*). Only in one case have we observed microscopically a thinning (*atrophy*) of the mucous membrane; but hypertrophy of the mucous membrane, which may become even five times as thick as it is normally, often occurs, especially in the warty condition, and in polyposis. The thickened tissue is, as a rule, compact, inelastic and fibrous. On being cut, it offers considerable resistance to the knife, and sometimes gives out a crackling sound, and the stiff edges of the incision do not retract. We can often see with our unaided eyes an increase of the submucous connective tissue and a thickening of the muscles, which are also hard to cut. In other cases of chronic gastric catarrh, dilatation of the stomach is caused by relaxation and thinning of the muscles, in which case the stomach increases to double, or treble its normal size. The remarks on ulcers of the stomach (p. 7) may be consulted with respect to ulceration in this chronic catarrh. The mucous membrane of the left sac may also become implicated in very severe cases.

2. *Condition of the intestine.* The morbid appearances in intestinal catarrh resemble those of the stomach. Here also we find that the mucous membrane sometimes has dark red streaks and spots, and at other times is of a slate-grey colour resembling that of an eel's skin; the vessels appear very sinuous and dilated; and the surface of the mucous membrane is covered with mucus which is ropy, grey, glairy or purulent, and sometimes lumpy. The follicles are at first swollen, and later on become atrophied and disappear, on account of which the mucous membrane assumes an areolated appearance. The contents of the small intestine may become very watery, and often resemble whey clouded with flakes. Although the mucous membrane is on rare occasions thin, it is generally considerably thickened, and is very compact and wrinkled in consequence of the proliferation of the interglandular and submucous connective tissue. In the small intestine we frequently find papillary and polypoid proliferations similar to those in the stomach. Contractions and dilations in consequence of, respectively, hypertrophy and atrophy of the intestinal muscles, are likewise met with in chronic catarrh.

In the large intestine, and especially in stomach-like dilatations of the colon, the follicles frequently take a very active part in the inflammatory process, and either project in the form of nodular swellings, which are of a mucous, purulent,

or thick pappy nature and vary in size from a grain of millet to a pea ; or they develop, by ulceration, into ulcers, which frequently cover a large portion of the large intestine. Röll states that they vary from the size of a hemp seed to that of a lentil ; that they extend as far down as the submucous connective tissue ; that they have sharp, non-infiltrated, undermined edges and a suppurating base ; that they frequently form large ulcers by confluence ; and that they either heal and leave behind a slight cicatrix, or cause perforation of the intestine, after having produced repeated diarrhoea and colic during a long period. (See "Intestinal Ulcers," p. 7).

**Symptoms.**—*Symptoms of chronic gastric catarrh.* The principal symptoms of a catarrh which is chiefly confined to the stomach, are, as in acute catarrh, loss and capriciousness of appetite, frequent yawning, delayed defecation, coated tongue, dry and slimy mouth, but generally no fever. These symptoms, which are often the only ones present at the beginning of an attack, usually undergo a remarkable change later on. The appetite is greatly disturbed after severe exertion, or even after ordinary work ; but may soon become more or less normal. Even slight faults in feeding may aggravate the condition, especially bringing on colic ; and, consequently, frequent attacks of colic are by no means rare in chronic gastric catarrh. In severe and long protracted cases we sometimes find peculiar nervous derangements, which were formerly comprised under the name of "stomach staggers." Besides debility and disinclination for work, which are always present, the animal may exhibit cerebral depression and even vertigo. Attempts to explain these mental disturbances have been made by ascribing the cause to conduction of the nervous irritation of the stomach to the brain ; anaemia of the brain produced by hyperaemia of the alimentary canal ; and absorption into the blood of abnormal substances formed in the affected stomach (auto-infection).

2. *Symptoms of chronic intestinal catarrh.* The symptoms vary greatly according to the intensity, duration and extent of the complaint. The abdomen is usually empty and tucked up, although at times it may be tympanitic ; the intestinal sounds vary a good deal in their nature, but are generally more or less suppressed ; and frequently constipation appears, the faeces being often formed into small balls, dry, with an acid or putrid smell, and sometimes remarkably pale and rich in un-

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digested food. In other cases we may find the chief symptom to be chronic diarrhoea, which will then indicate the presence of chronic intestinal catarrh. Frequently constipation and diarrhoea occur alternately. We may almost always observe flatulence and frequently recurring signs of uneasiness. Long-standing cases are marked by mal-nutrition; the animal becomes weak, emaciated and sweats readily; the mucous membranes are pale or yellow; the hair is staring and lustreless; the pulse, small and weak, and the action of the heart irregular and often intermittent. The urine affords no constant indication, and in some cases is quite normal. As in acute intestinal catarrh, its colour varies greatly and it is frequently slimy. In most cases its reaction is alkaline, which statement is confirmed by Albrecht; but Siedamgrotzky and Hofmeister report that they have almost invariably found it acid. Occasionally the urine gives a slight acid reaction, which may be attributed to exacerbations of the chronic process, or to a special affection of the small intestine. In a few cases we have found large quantities of albumen in the urine.

**Duration and Course.**—The duration depends on the possibility of removing the causes, and on the extent and intensity of the disease. The more easily the cause can be recognised, the quicker can it be removed, and the shorter and more favourable will be the course of the malady. This remark is specially applicable to catarrh caused by faults in feeding. Severe lesions in the mucous membrane will, however, delay recovery. Horses which suffer from chronic catarrh of the stomach frequently continue at work for years, while constantly undergoing alternative improvement and exacerbation, although they gradually lose their strength and weight under such a condition. As a rule, such animals are brought to veterinary surgeons for treatment, not on account of the chronic disease, but in consequence of the recurring exacerbations. In other cases total incapacity for work is caused by increasing weakness, and death from exhaustion soon follows. It is probable that the majority of old and decrepit horses perish in this manner. If the chronic catarrh has brought about dilatation of the stomach, death may sometimes occur suddenly from colic.

**Treatment.**—The principles of treatment are essentially the same as those for acute catarrh (p. 59). Attention should

be chiefly paid to removal of the causes and to proper feeding. We should give digestible food in small and frequent quantities. For instance, we may add an extra meal in the forenoon and afternoon, and we should regulate the diet according to the work. Any sharp points of teeth should be removed, and the oats given to old animals should be bruised. Owners have often extreme difficulty in establishing a proper method of feeding in their stables.

Medical treatment is only of secondary importance. Hydrochloric acid in small doses (2 to 3 drams), has also proved itself here to be very efficacious. The principal part in the therapeutics of this disease is, however, played by alkalies, as is acknowledged by all practitioners. For instance, we may give a tablespoonful of common salt in each feed for some time, and bicarbonate of soda and Glauber's salts in doses of  $\frac{1}{2}$  oz. to  $1\frac{1}{2}$  oz. The simultaneous administration of the above-mentioned three salts in the form of artificial Carlsbad salt is particularly efficacious. Carlsbad salt in doses of  $\frac{1}{2}$  oz. to  $1\frac{1}{2}$  oz. neutralises acidity; stimulates, by its contained salt, the secretion of the digestive fluids; prevents fermentation; dissolvesropy mucus, and by its contained sulphate of soda causes evacuation of the feces. As in acute catarrh of the stomach, we may also use rhubarb, gentian, aloes, pepsin and apomorphine. For constipation we may employ green food and mashes; comparatively large doses of Glauber's salts ( $\frac{1}{2}$  to 1lb.); medium doses of aloes; and enemas of cold water, soap and water, glycerine, etc. Kattner, Schindelka and others recommend the injection of glycerine (3 to 6 oz.) into the rectum by means of a syringe having a long tube. It has a very irritating effect on the mucous membrane of the large intestine and produces peristalsis and defecation by reflex action. We have found that enemas of glycerine have about the same effect as those of soap and water, the action of both being simply to clear out the rectum. They have no power to remove fecal accumulations which are situated higher up in the intestine, and are consequently of no use in colic caused by impaction of food.

For diarrhoea we may use the astringent and styptic remedies recommended for acute catarrh. Opium is in all cases the most reliable agent. Styptic agents in the form of enemas are useful only in catarrh of the rectum, beyond which they cannot be forced under ordinary conditions. According to Albrecht, creolin (2 to 3 drams daily) is the best remedy for

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chronic gastro-intestinal catarrh. By its action the appetite and digestion quickly improve, peristalsis becomes stimulated, and the faeces almost immediately lose their bad odour.

### ACUTE GASTRO-INTESTINAL CATARRH OF CATTLE.

(*Acute bovine dyspepsia.*)

**General Remarks on the Diseases of the Gastro-intestinal Canal of Ruminants.**—The knowledge which we possess respecting the diseases of the organs of digestion of ruminants is so incomplete, that veterinary surgeons are obliged to include totally different pathological conditions of the stomach and intestines of cattle under the name of indigestion. Our ignorance in this case is due to the complicated nature of the stomach of ruminants, the four compartments of which are entirely different from each other in arrangement and function, and thus, instead of having only one disease, as in the horse and dog, we may have four collateral ones.

With cattle, as with horses, we have preferred to discuss so-called acute dyspepsia along with acute gastro-intestinal catarrh, and not as an independent disease. It appears that in the beginning of many bovine cases the alteration is in the function and not in the structure of the implicated organ, which statement is all the more probable, because the mucous membrane of the first three stomachs is very resistant to a catarrhal affection. For instance, rumination may be entirely suppressed merely on account of nervous or motor disturbances of the rumen, without any previous inflammation of its mucous membrane. As these anomalies of function, when they are not altogether temporary, are directly followed by acute catarrh, as the causes of dyspepsia and catarrh are the same, and as their respective differentiating symptoms cannot be distinguished from each other, we have thought it best, contrary to usual custom, to describe acute dyspepsia only as the first stage of acute gastro-intestinal catarrh.

In conformity with the complicated structure of the stomach of ruminants, we find that the three ante-stomachs are chiefly attacked, and especially the rumen and omasum. In some cases the abomasum and duodenum are prominently implicated. Usually the intestine is involved only sympathetically, and its affection offers, externally, few characteristic symptoms, although sometimes even the affection of the intestine may

become very prominent, in which case diarrhoea will form the chief symptom.

**Etiology.**—Individual susceptibility to acute gastro-intestinal catarrh varies a good deal, but its causes are not well known. Frequently they can be attributed to weakness of digestion, relaxed constitution, previous disease, age, etc. The principal pre-disposing causes are errors of feeding, such as the practice of giving relaxing food without sufficient rough fodder, or forcing the milk and, later on, fattening quickly. In small byres, belonging to poor people, especially near towns and in times of scarcity, animals are often fed on any collected refuse. In consequence of the unnatural method of feeding and housing, cattle in certain places (cows in particular) become affected more readily than others. The disease is more rife during spring and autumn than at other times. The following are the chief direct causes :—

1. Overfeeding.
2. Fodder which is frozen or covered with hoar frost ; pasturing too early in the spring and too late in autumn ; unaccustomed cold drinks ; chills and too hot food.
3. Improper feeding, such as abrupt changes from dry to green food, and *vice versa* ; too new hay, especially if the animals soon afterwards drink large quantities of water ; excessive feeding on straw, bran, pollards, roots, potatoes, oilcakes, unbruised grain, chaff, bye-products from breweries and distilleries ; admixture of weeds, and turnip tops ; sudden changes to unaccustomed food ; fattening on dry food, etc. Although feeding on short-cut straw is generally regarded as injurious, the experiments made by Friedberger do not unconditionally confirm that idea.
4. Spoilt and decomposing food, such as kitchen refuse ; fermenting and acid refuse from factories ; hay which has been exposed to inundations, etc.
5. Indigestible food, such as tough, lignified plants ; green flax ; foreign bodies, especially sand and gravel ; the eating of the after-birth by cows, etc.
6. Too severe and too long continued exertion, which causes rumination to cease. We may point out that hard though well regulated draught-work, which the oxen of millers, brewers, farmers and others on the Continent have to do, causes no disturbance of digestion.

**Symptoms.**—I. *The dyspeptic stage of acute gastro-intes-*

*tinal catarrh.* Frequently the initial symptoms of this disease are restricted to disturbances of appetite and digestion, in which case the animal loses its zest for food, prefers green stuff to dry fodder, and fresh spring water to its usual drink, or it may begin to feed greedily and then soon stop eating; it lies down a good deal, becomes depressed, and ceases to lick out its nostrils with its tongue; rumination becomes infrequent and shortened in duration; the movements of the paunch become suppressed or slow; defecation is somewhat delayed, and the faeces are normal or inclined to be dry. There is no fever, the extremities are warm, the muzzle is cool and moist, and the secretion of milk but little diminished. If the morbid condition does not pass off in, say, four to five days, it will develop true catarrh.

2. *True acute catarrh of the stomach and intestines* (which either commences as such, or develops from the condition described in the preceding paragraph). It would be difficult to sketch a typical picture of the disease, and we shall therefore content ourselves by briefly noting the usual symptoms, which are as follows:—The animal is depressed and stands back almost constantly from the feeding trough; he arches his back and places his feet under the body (like a chamois); the coat stands on end, especially on the head, over the eyebrows, and along the spine; the ears hang down or lie back; the temperature of the surface varies, particularly that of the ears and base of the horns, and occasionally there are slight rigors. The temperature of the muzzle is also variable; it still remains moist, though to a less extent than in its normal condition, and if the beads of moisture on it be wiped off, a considerable space of time will pass before they are replaced. Usually the conjunctiva is highly injected.

The oral cavity is abnormally warm; the mucus and saliva contained in it are increased in amount, and even salivation is sometimes observed; appetite and thirst are greatly diminished or entirely suppressed, and rumination generally ceases; even if it continue to exist, it proceeds slowly and in a languid manner; the morsels are small, and each bolus receives only about half of the usual number of grinding movements; we may occasionally notice eructations of stinking gases and even vomiting. As a rule the abdomen is distended, particularly on the left side, both above and below, and the left flank abnormally prominent. The contents of the paunch may be felt at the flank as a consistent dough-like mass, but if the wall

of the flank is puffed out with gas, which has collected in the upper portion of the abdomen, we can feel the semi-solid contents only by energetic palpation, in which case a dull knocking sound is heard. Usually there is a temporary change from time to time in the fulness of the left flank, in consequence of the oft recurring inflation. The movements and sounds of the rumen are reduced, although its contents are often heaved up, but they are not rotated as much as during health. Pressure on the left flank and hypochondrium does not appear to be painful.

Defecation is usually retarded. The dung is generally more solid and darker in colour than normal—annulated, sometimes stinking, and may be covered with a thin crust, or it may be shining, pasty and surrounded with mucus. If thin diarrhoea comes on, we shall often find in it particles of entirely undigested food. By auscultation of the right wall of the abdomen, we may hear rumbling and rattling intestinal murmurs. Besides these symptoms, the animal is often somewhat restless, knuckles over with the hind feet, kicks at his belly, whisks his tail, repeatedly lies down and gets up, looks round at his hind quarters, assumes a lateral recumbent position, etc. In other cases severe constipation may ensue. The urine, which at the beginning of the attack was alkaline and of a bright sherry-yellow colour, becomes scanty later on, dark in colour and acid. The milk often decreases rapidly down to as little as half its usual quantity.

Fever is generally present. The pulse, as a rule, is small, hard and accelerated by from 10 to 20 beats per minute. The temperature of the rectum is increased, though not always in a uniform manner, on which account, as Harms has justly pointed out, we cannot always judge with certainty the severity of the malady from the state of the internal temperature. Moreover, we should bear in mind that in cattle the pulse and temperature vary a good deal, even under normal conditions. We shall, therefore, do well to feel the pulse and take the temperature of some of the healthy animals of the same herd or byre.

**Process and Termination.**—In mild cases, which are the rule, improvement begins in from five to eight days, the movements of the paunch become more frequent, and its sounds more distinct; rumination and appetite gradually reappear; defecation becomes more normal; the secretion of milk increases,

and fever gradually abates. In severe cases, on the contrary, the symptoms become intensified; the mouth is dry, coated, hot and emits a bad smell; rumination, consumption of food and the movements of the paunch are suppressed; either constipation or diarrhoea is present; milk secretion is arrested and the udder is flabby and relaxed; the movements are feeble, the sensory apparatus depressed, and the animal utters faint groans and sighs when lying down. The expression is plaintive; the eyes are sunken; mucus resembling sago accumulates at the inner angle of the eye; the fever rises to  $40^{\circ}$  C. ( $104^{\circ}$  F.), or more, and the rate of the pulse is increased by 30 or 40 beats a minute. The ears and horns are alternatively hot and cold; the muzzle remains continually hot and dry; and respiration, which was quick from the beginning, becomes painful and still more accelerated. With these symptoms the disease approaches in character inflammation of the stomach and intestines, which usually ends in death. If acute catarrh runs a long course, it may pass into the chronic form.

**Differential Diagnosis.**—The symptoms, duration, course and termination of acute gastro-intestinal catarrh of cattle are, on the whole, so characteristic, that its diagnosis is easy. It is of course often impossible to say which part of the alimentary canal is chiefly affected; but this is not of much importance, because, as a rule, the affection quickly spreads from its first site to the neighbouring parts. The accumulation of gas in the rumen might cause acute catarrh to be mistaken for tympanites; but the slight amount of the flatulence, and its periodic and varying nature furnish ample data for differentiation. It is often very difficult to distinguish severe acute catarrh from inflammation of the stomach and intestines, especially as one may develop into the other.

**Prognosis.**—Recovery is nearly certain in simple catarrh. Hering states that in 128 cases of "indigestion," only five were slaughtered, and that resort to this measure was not absolutely necessary. Acute catarrh may, however, develop into chronic catarrh, which is more dangerous to life than the acute form, and may even run into inflammation. Consequently, we should make our prognosis at the beginning of the attack with a certain amount of reserve.

**Anatomy.**—Owing to the fact that very few *post-mortem*

examinations are made on account of the small percentage of deaths, our knowledge of the pathological anatomy of the disease is small. Harms states that the mucous membrane is generally swollen, congested in stripes, in spots, or diffusely, and that, especially in the abomasum and intestine, it is covered with a coating rich in cells. The catarrhal symptoms are best marked on those parts of the rumen which possess large papillæ, and are less evident in the reticulum and omasum, of which a few leaves are now and then severely affected. Some authorities state that the contents of the omasum are in places quite dry, and of a coarse hard nature in the vicinity of the free edges of the large laminae.

**Treatment.**—The most important point is to observe a strictly hygienic method of feeding. So long as rumination is suppressed, we should keep the animal more or less fasting, and give it nothing except a very little easily digestible food, as, for instance, freshly-mown meadow grass, tender hay or aftermath, more with a view to stimulate the movements of the stomach, than to satisfy any appetite which might be present. The animal should be kept on short diet, even after rumination has become re-established, because active treatment cannot be applied until the rumen is freed from the masses of food which have accumulated in it. At the same time we should encourage thirst by giving salted gruel or whey. If these are refused, we may gain our object by getting the patient frequently to lick salt. The movements of the stomach and intestines may be stimulated by hand-rubbing the surface of the trunk, and especially over the region of the paunch from the left flank. If defecation be delayed, the animal should be frequently back-raked, and enemas of soap and water or salt and water employed.

If simple dyspeptic symptoms exist, we may give internally 2 or 3 drams of hydrochloric acid two or three times daily in a bottle of water. If at the same time there is constipation, as is usually the case, we may give a mild laxative, such as sulphate of magnesia or Glauber's salts ( $1\frac{1}{2}$  lbs.), dissolved in two or three quarts of linseed tea, and divided into three doses to be given at intervals of about three hours. For retarded or suppressed rumination tartar emetic and hellebore are the best stimulants, and either of them may be given in doses of 2 to 4 drams; or we may inject subcutaneously  $1\frac{1}{2}$  to 2 grains of veratrine, dissolved in  $\frac{1}{2}$  oz. of 2% alcohol.

Some advise creolin in doses of from  $\frac{1}{2}$  to 1 oz. Sulphate of physostigmine has been employed with good results for atony and paralysis of the stomach and intestine. Feser, as the result of his experiments with cattle of the smaller breeds, recommends  $1\frac{1}{2}$  grains of it dissolved in 40 minims of water, to be injected under the skin; and it may be repeated in five or six hours if the desired result has not been attained. Eber considers that the best agent is eseridinum tartaricum ( $2\frac{1}{2}$  to  $4\frac{1}{2}$  grains). Under certain conditions, puncture of the rumen and the use of a probang may be necessary.

#### ACUTE GASTRO-INTESTINAL CATARRH OF SHEEP.

In this disease we find the same etiological and symptomatic conditions as in the bovine form. Chill and errors of feeding are their chief causes. Gerlach observed gastro-intestinal catarrh in flocks of sheep which had been pastured on luxuriant meadows; and Schick had a similar experience with sheep that had been fed on large quantities of raw potatoes. Here also the chief symptoms are: diminished consumption of food; disturbances of rumination; thin and even watery diarrhoea; occasional constipation; faintness, etc. As compared with cattle, sheep are much more liable to succumb to this disease, in consequence of their weaker constitution. Schick reports that in a herd of 600 sheep, 60 died within a week; and Gerlach, that in a herd of 300 sheep, 20 were dead in four days. On *post mortem* examination, the changes were found to be similar to those produced "by severe cathartics." Some consider it to be enzootic (an infective disease), on account of the fact that it often spreads through entire flocks.

#### CHRONIC GASTRO-INTESTINAL CATARRH IN CATTLE.

(*Chronic dyspepsia or Omasitis chronica.*)

**General Remarks.**—Chronic gastro-intestinal catarrh of cattle is generally described under the name of "impaction of the omasum." This designation is due to the fact that the accumulation of firm, dry masses of food in the omasum was always considered to be the chief morbid aspect of the disease, which was never diagnosed as a chronic catarrh of the stomach or intestine, as in other animals, but always as an omasitis, namely, so-called impaction of the omasum. This name is by no means distinctive, as has been pointed out for a long time by many observers. In the first place, such an accumulation of food in

the omasum occurs in various bovine maladies, during the course of which, appetite, rumination and the movements of the paunch are for a long time suppressed, and the contents of the omasum become gradually dry from absence of saliva, or from absorption of the fluid. The condition in question is met with in all severe febrile diseases, and particularly in digestive troubles. For instance, rinderpest was formerly called "dry omasum." Furthermore, a true obstruction to the passage of food through the omasum hardly ever occurs. Harms has observed only one case. The old name should therefore be given up:

There are, however, cases in which the catarrh may become prominently localised in the omasum, but they are rarer than the chronic affections of the paunch, abomasum and intestine. Catarrh of the omasum is generally symptomatic of a disease of the rumen, etc. As it is impossible to give a separate description of the respective catarrhs of the intestine and of each of the four stomachs, we have comprised the chronic inflammatory condition of the mucous membrane of these divisions of the alimentary canal under one heading, namely, "chronic gastro-intestinal catarrh."

**Etiology.**—All the causes of acute catarrh described may produce chronic catarrh, provided that they are more continuous or more intense. Neglected or irrational treatment of acute catarrh frequently induces chronic catarrh. We have also to consider the following factors :—

1. Imperfect mastication, owing to defective teeth or absence of teeth.
2. Adhesion of the rumen or reticulum to the abdominal wall, in consequence of circumscribed peritonitis.
3. Compression of the oesophagus by new growths, and consequent impediment to rumination.
4. New growths in the various compartments of the stomach, as, for instance, scirrhus and sarcomata in the abomasum; papillary proliferations at the opening of the reticulum and omasum; and papillomata and armour-like sarcomata, etc.
5. Displacement of the reticulum by rupture of the dia-phragm; diaphragmatic hernia of the reticulum.
6. Foreign bodies in the stomach and intestines, such as nails, and balls of hair, plants and wool; new growths; diverticula and stenoses in the intestine; and intestinal vesicular fistulae.

7. Vascular engorgement in chronic diseases of the liver, lungs and heart.
8. Pressure of the gravid uterus on the rumen and weakness of the movements of the rumen.
9. Chronic gastro-intestinal catarrh is found secondarily in all chronic disturbances of nutrition, especially in tuberculosis.

**Symptoms.**—The symptoms of chronic catarrh differ but little from the acute form, except as regards their longer duration and greater persistency. The disease is characterised by frequent remissions and exacerbations. The general health is upset; the animal is depressed and feverish; the temperature of the body is unequally distributed; the skin hidebound; the coat rough and lustreless; gradually increasing emaciation appears; the mouth shows changes similar to those described in acute catarrh; consumption of food, rumination and the movements of the stomach are diminished or entirely stopped; signs of pica (p. 47) are sometimes present; eructations are rare, but when they occur, are foetid; the paunch is usually hard, firm to the touch and considerably filled with food; when the flanks are sharply tapped a distinct knocking sound is heard. We can generally observe slight inflation of the rumen, which varies according to the consumption of food, and becomes continuous after the movement of the stomach has ceased. Vomiting has been noticed in a few cases.

Defecation is considerably delayed, and the scanty dung is passed in the form of dry, peat-like, dark-coloured balls, which often contain many particles of imperfectly masticated and undigested food, and are generally covered with tenacious, glairy or bloody mucus, sometimes in shreds. At other times soft, thin, acid or stinking masses are passed, in which case the diarrhoea will last for weeks, if not months, and may lead to death from exhaustion. Usually constipation and diarrhoea alternate. The quantity of urine is generally small, and is proportionate to the decreased consumption of water. We have no exact statements as to its reaction. Milk secretion is diminished or entirely arrested. Groaning and grinding of the teeth are often observed.

If recovery does not set in, the intensity of the symptoms, as a rule, will quickly increase. The appetite is lost; the mucus becomes dry and fissured; the fever higher; the pulse small and weak; the heart palpitating; tympany increases; the movements of the stomach are entirely suspended; complete

constipation or persistent diarrhoea sets in, and emaciation increases. The animal is very weak and prostrate ; lies down a great deal, groans uninterruptedly, and is eventually incapable of getting up. The expression is plaintive, the eyes sunken, and death takes place, either naturally, or by slaughter.

**Anatomy.**—The anatomical changes vary greatly, according to the localisation, intensity and duration of the chronic catarrh.

The *rumen* is somewhat inflated by gas, and contains a great quantity of dry food. The epithelium of the mucous membrane can often be readily detached soon after death, although in a healthy carcase it remains firmly adherent for a long time. According to Bruchmüller, it is occasionally thickened. The mucous membrane, especially in the part which has large papillæ, is interspersed with ecchymoses, either uniformly or in spots, and is occasionally swollen, thickened and of a purple or brown-red colour. The papillæ may be either in an atrophied or hypertrophied condition.

The *reticulum* is usually least affected, and sometimes contains a large quantity of dried-up coarse food. Its mucous membrane has appearances similar to those in the rumen.

The *omasum* is considerably dilated and completely filled with food, the cakes between its leaves being compressed and unusually dry. The epithelium of the mucous membrane is also abnormally detachable, and often adheres to the dry cakes. The ingesta is either coarser than in the rumen, or is finer, because it has been ruminated, and can be broken up between the fingers into a fine granular powder. The mucous membrane of the leaves may be congested diffusely, in spots, or in streaks ; may be inflamed, soft and tender ; gangrenous, or interspersed with dark-red foci and haemorrhagic ulcers.

The *abomasum*, as a rule, is more or less empty, and often contains coarse chyme. Its mucous membrane shows an intense infiltration of pigment in the form of slate-grey streaks and spots. It is also thin and smooth (atrophied) and covered with a tenacious, glairy fluid.

The *intestinal canal* shows various changes. Its mucous membrane may be markedly congested, changed in colour to slate-grey, atrophied, and cribiform and pitted, in consequence of the obliteration of the Peyer's patches. The contents of the *duodenum* usually consist of large quantities of bile-coloured mucous masses. In the *large intestine* we generally find dry,

dark coloured faeces, and the mucous membrane covered with a thick coating. When chronic catarrh turns into phlegmonous inflammation of the intestine, or into peritonitis, we have the respective changes of the two last-mentioned diseases. The gall-bladder is generally filled with thick bile. The liver shows various stages of engorgement.

The other conditions vary greatly, and may usually be referred to venous congestion. They consist of hyperæmia of the lungs, meninges of the brain, kidneys, right side of the heart, venous system, etc.

**Differential Diagnosis.**—Owing to its very varied aspect, chronic gastro-intestinal catarrh may be confused with several diseases, such as acute catarrh and acute phlegmonous inflammation of the intestines; but its chronic course will be its characteristic sign in both these cases. It may often be very difficult to distinguish it from chronic peritonitis, brought on perhaps by a perforating foreign body, because this peritonitis frequently becomes complicated with chronic catarrh, and the peritoneum may become sympathetically affected by an intense catarrh. In many cases we obtain an explanation of the nature of the malady only by the course of the disease (as by supervening symptoms of heart disease), or *post mortem*. A large accumulation of gas in the paunch being the chief symptom of acute or chronic tympanites, may suggest either of these diseases, because meteorism occurs but occasionally and only to a slight degree in chronic catarrh. Finally, chronic gastro-intestinal catarrh may be confounded with tuberculosis, which is a chronic disease that shows emaciation, fever, disturbances of digestion and nutrition, etc. An examination of the lungs, and the use of tuberculin will enable us to make a prompt and exact diagnosis.

**Prognosis.**—It is well to exercise caution in making a favourable prognosis in the beginning of the attack. There is small chance of recovery if the disease lasts longer than two or three weeks; if complete constipation be present for several days; if diarrhoea persists for a week or longer; or when the appetite, rumination and the movements of the intestine remain entirely suppressed. Slaughter will then be generally preferable to treatment.

**Treatment.**—The therapeutic measures for acute gastro-

intestinal catarrh also hold good for chronic catarrh. Thus, in benign dyspeptic cases, besides attending to diet, it is advisable to give hydrochloric acid several times a day in doses of 2 drams. Small doses of common salt, bicarbonate of soda, Glauber's salts, or artificial Carlsbad salts (3 to 10 ozs.), continued for weeks, have sometimes a good result. If the ante-stomachs are greatly relaxed, and if inflammatory symptoms have not yet appeared, we may give oil of turpentine (two or three times a day, in doses of 2 to 6 drams) in aromatic vegetable infusions; spirits of wine (1 to 2 ozs.), powdered hellebore root, and veratrine subcutaneously. Truelsen finds that creolin (1 to 2 drams in a bottle of water, three times daily) is the best remedy, and that, with it, recovery takes place in a few days. As aperients we can recommend large doses of Glauber's salts (16 to 32 ozs.), or Carlsbad salts (p. 66) with large quantities of water; castor oil (1 to 2 pints); aloes (1 to 2 ozs.); and in cases of very obstinate constipation, tartar emetic ( $\frac{1}{2}$  to 5 dr.), or calomel (15 to 75 grains *pro die*), which should be used cautiously. The much-lauded sulphate of eserine (1 gr. subcutaneously) appears by recent observations to be occasionally ineffective, and sometimes to cause abortion, dangerous dyspepsia, suffocation, etc. If slaughter be probable, care should be taken not to use drugs that would spoil the meat. Höhne and others have obtained many recoveries by filling the rumen with large quantities of warm water, or salt and water, by means of a trocar and funnel. Deffke injects eleven gallons of lukewarm water within a few hours, and allows the canula to remain in all the time. He praises the good effect obtained from an addition of hydrochloric acid ( $\frac{1}{2}$  dr. to 26 pints of water), and the injection of an infusion of valerian root, and advises that the injection should not be delayed too long. We may give raw linseed oil and frequent enemas of water.

If the rumen be filled with very firm masses of food, and medicines have proved useless, and there is a probability of foreign bodies being present in the stomach, we may open the rumen as a last resource, and remove a part of the contents.

#### ACUTE TYMPANITES OF RUMINANTS.

**Nature.**—In this disease the distension of the rumen by gas is far greater than in chronic tympanites. It is seen most frequently in cattle, and next to them in sheep. Many animals, especially sheep, are often affected at the same time.

**Etiology.**—*General causes.* The chief cause is the introduction into the stomach of a large amount of food which ferments and produces great quantities of gases. Tympain is caused most frequently by green food, especially when it has begun to ferment, as may happen in stalls. This fact explains the usual occurrence of this malady on holidays, particularly when there is a succession of these days, and a large supply of green food has been laid in. We also know from experience that this affection is most frequent during damp, warm weather, which stimulates fermentation. It may also be caused by a sudden change from dry fodder to green food; by feeding on grass which is rank, luxuriant, wet or frozen; and by allowing cattle to drink too soon after eating.

Some animals are predisposed to tympanites by the habit of eating too quickly or too immoderately, or by weakness in the digestive organs, which would, however, predispose more to chronic tympain than to the acute form.

*Special causes.*—Here we have to consider chiefly the action of certain food-plants, among which the following are specially liable to produce an attack:—

1. Clovers, such as lucerne (*medicago sativa*), sainfoin (*onobrychis sativa*), and particularly red clover (*trifolium pratense*). Clover is much more dangerous before blossoming than after it has become mature, and, probably, when the luxuriance of its growth has been promoted by manuring it with gypsum.

2. Other leguminous plants, such as green lentils or vetches and buckwheat.

3. Grasses grown on wet soil; reed grasses, such as *poa aquatica* and *scirpus sylvaticus*, which are covered with microbes; potato and turnip tops; leaves of cabbage and rape; charlock (*sinapis arvensis*) and wild radish (*raphanus raphanistrum*).

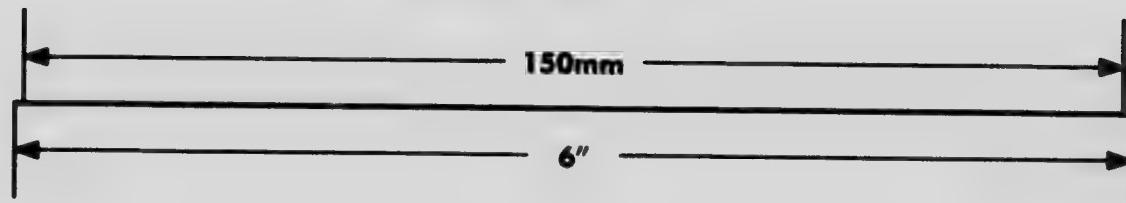
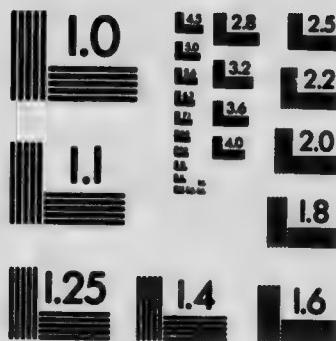
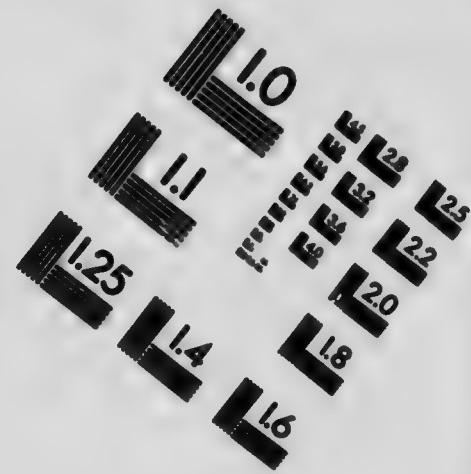
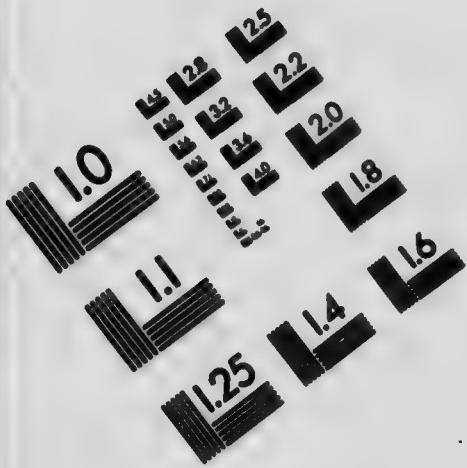
4. Green corn; feeding on stubbles containing blades of corn and weeds. Plants grown in the dark are more dangerous than when they have been exposed for some time to sunlight.

5. Fermenting drinks, brewers' by-products, rotten potatoes and turnips, cotton-seed meal and other articles, especially when the animal has not been accustomed to them, and when they are eaten hastily and in large quantities.

6. Tympanites may occur as a symptom of various poisonings, such as by meadow saffron (*cochicum autumnale*), hemlock (*conium maculatum*), water hemlock (*cicuta virosa*), deadly nightshade (*atropa belladonna*), yew (*taxus baccata*), white hellebore (*veratrum album*), common red poppy (*papaver rhoeas*),

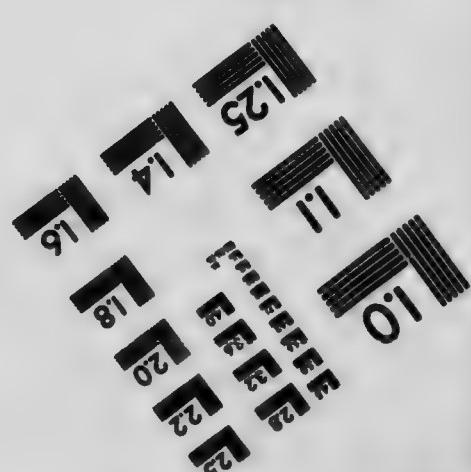
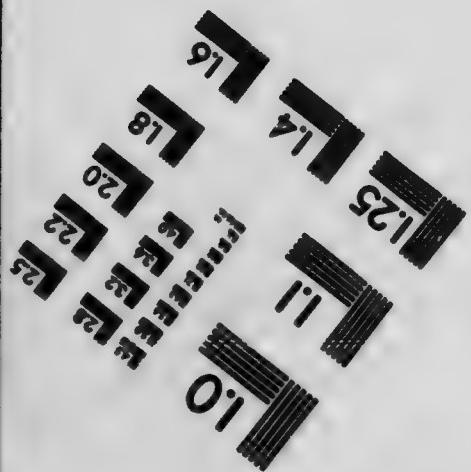


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tobacco, various kinds of ranunculus, mould fungi, salt fish and salt-meat brine, etc.

7. Cattle seem sometimes to blow themselves out in a manner analogous to the wind-sucking of horses. Calves also, at times, swallow an inordinate quantity of air when sucking the teat or other object.

8. Foreign bodies which have stuck in the oesophagus may produce tympanites by impeding eructation. We met a case of this kind which was caused by a hairball that had been thrown up into the gullet from the stomach.

*Post-mortem appearances.*—The contents of the rumen, besides food and especially grass, consist chiefly of carbonic acid, carburetted hydrogen, hydrogen, sulphuretted hydrogen, oxygen and nitrogen. Carbonic acid is in abundance when the animal has been fed on green food, and carburetted hydrogen when the forage has been dry. Reiset found that the contents of the rumen of a cow which had died from tympanites two hours after feeding in a field of clover was as follows :

Carbonic acid .. ..	74 per cent.
Carburetted hydrogen .. ..	24 .. ..
Nitrogen .. ..	2 .. ..

In a tympanitic wether, Reiset found 76 per cent. of carbonic acid, but no sulphuretted hydrogen, the presence of which appears to be accidental, because it is a physiological product of the decomposition of albumen in the rumen.

Excessive gaseous distension may cause rupture of the paunch and also of the diaphragm, and, consequently, escape of gas and food into the abdominal cavity, which seldom leaves signs of peritonitis, owing to the rapidly fatal course of the affection. The superficial, subcutaneous and large veins (jugular and axillary) are gorged with blood, which is dark red, thick, sticky, coagulates badly, and assumes a bright red colour in the presence of air. The lungs are very hyperæmic, and are interspersed with subpleural and parenchymatous haemorrhages. Pulmonary œdema is occasionally present. We also find haemorrhages under the costal portion of the pleuræ, especially in the upper and anterior regions. The right side of the heart is considerably filled with blood; the coronary veins are injected, and haemorrhages are found under the epicardium and endocardium. Rupture of the right wall of the heart occurs in rare cases. In the intestinal canal we find hyperæmia of the mucous membrane and submucous and sub-

## ACUTE TYMPANITES OF RUMINANTS.

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peritoneal haemorrhages. Rupture of the vessels of the spleen and destruction of its tissue have been observed. The mucous membranes of the head, cerebral membranes and sinuses are very hyperemic, and apoplectic foci are sometimes found in the brain.

**Symptoms.**—The most striking symptom is the sudden and rapidly developing increase in the size of the abdomen, especially in the left flank (position of the rumen), which is more or less prominent, in very severe cases its highest point being level with, or even projecting above, the spinal column. The abdominal walls are tense and very elastic to the touch, and percussion produces a full, clear, tympanitic sound, which may be even metallic. There are no normal sounds of the rumen. From the beginning of the attack appetite and rumination are suspended and, despite severe straining of the animal, defecation ceases after small quantities of dung have been repeatedly passed. The animal stands with the legs either placed under the body or spread out; the back is frequently arched; the tail raised, the ears hang down in a limp condition, and the animal can be made to move only by considerable urging.

With increasing tympany, the breathing becomes more accelerated and laborious; the animal grows anxious, excited, and totters hither and thither; the visible mucous membranes of the head, and particularly the conjunctiva and the vessels of the sclerotic, are greatly injected; the superficial veins, especially of the head and throat, and the milk veins are engorged and very prominent; the eyes stare; sweat breaks out at the base of the ears, behind the elbows and on the flanks; the pulse becomes more and more frequent and wiry (small and hard), and finally imperceptible; the heart-beat more palpitating; dyspnoea increases, and respiration is short and quick, while the nostrils are distended, and even the mouth is kept open and the tongue stretched out. The animal groans, moans, slavers, froths at the mouth, sometimes coughs slightly, stretches itself out, and manages by great exertion to make a few short and interrupted eructations. In rare cases vomiting occurs. The limbs become quite cold; the mucous membranes cyanotic (of a dark-blue or leaden colour); the patient stands stupefied and motionless, gradually begins to stagger, threatens to break down behind, falls, becomes convulsed and soon dies.

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**Pathogenesis.**—The symptoms are easily explained. Speaking generally, they are due to poisoning by carbonic acid, and in rare cases to cerebral apoplexy. The carbonic acid poisoning is partly produced by compression of the lungs caused by the forward displacement of the diaphragm, and partly by the blood absorbing that gas from the stomach. The congestion in the anterior half of the body (injection of the mucous membranes, hyperæmia of the brain, and venous congestion), arises from pressure of the tensely filled rumen against the large vascular trunks of the posterior part of the body, and from impediment to the return of the blood to the heart and lungs. Cerebral apoplexy and the formation of ecchymoses may result from excessive engorgement.

**Course and Prognosis.**—The course of acute tympanites is very rapid. In severe cases the animal may die in a few hours, and even in half an hour, if help be not speedily procured. A perfect cure is not always obtained by the mere removal of the accumulated gases, because the fermenting food may continue to generate fresh supplies for 12 to 24 hours. If the inflation be moderate, a spontaneous recovery may sometimes take place, in consequence of abundant eructations and intestinal evacuations. Prognosis is not very favourable without proper and early treatment, which is not always possible, considering the rapidity of the course.

**Treatment.**—As regards prophylaxis, we should exercise care in changing from dry to green food. At first we should avoid luxuriant pastures and, before taking cattle out, should give them some dry food, so that they may not begin to eat green stuff on an empty stomach; should keep them only for a short time on clover lands, and watch for any appearance of tympanites. Precautions should be taken to prevent cut green stuff from fermenting. If practicable, an oesophageal tube, trocar and canula should be kept in readiness in case of accident.

In almost all cases the removal of the gases is the first consideration. Among rough and always available methods of treatment, we may mention pulling the tongue repeatedly out of the mouth, by which means eructation is produced; and drawing a knotted straw band to and fro in the mouth, which brings on eructation, and sometimes even vomiting. A similar effect is produced by gagging the animal with a straw rope smeared with cart-grease, tar or other nauseous stuff,

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and tied behind its horns. For the same purpose, butter or lard (cattle  $\frac{1}{2}$  lb. and sheep 1 oz.) is greatly used in some parts of France. Exercise may do good, but it is not without danger.

Rychner justly calls strong and steady pressure on the left flank the "safest," "cheapest," "most convenient," and, we might add, "most harmless" means for reducing inflation, especially in mild cases. In performing this operation, we can feel how the pressure of the hand stimulates the movements of the rumen, after the restoration of which eructation often quickly takes place. We can completely empty the paunch of gas by this method of long continued pressure, and by applying massage with both hands to the abdomen.

As a rule, pressure and massage will effect a cure. Brandy ( $\frac{1}{2}$  to 1 pint in double the quantity of cold water) and tincture of veratrine ( $\frac{1}{2}$  oz. in alcohol), are probably the best medicinal means. We might also try oil of turpentine ( $1\frac{1}{2}$  to 4 oz. in weak spirits and water or oil), which is highly recommended by some authorities, although it fails as often as it succeeds. Assafoetida, ether, paraffin oil, camphor, eserine and ammonia should not be used.

On approaching danger, we should resort without delay to the employment of the oesophageal tube and to puncture of the rumen. The introduction of this tube cannot always be managed without risk, especially when the animal is very restless and suffers from great dyspnoea. The escape of gas is sometimes prevented by the tube getting plugged up with food, in which case nothing is left but puncture of the rumen. A previous incision of the skin will greatly facilitate this operation. The best kind of trocar is a round one, without lateral openings in the canula. The escape of the gases should be stopped from time to time, so that anaemia of the brain and consequent swooning may not be produced by hyperæmia of the rumen from diminution of pressure. The trocar should not be taken out too soon, because the continued fermentation of the contents of the rumen may set up renewed inflation. The canula, which is closed with a cork, should be allowed to remain in for some hours longer, during which time it should be continuously watched, so that it may not fall out. In default of a trocar, the operation may be performed with a knife. After the primary danger has been averted, it is imperative to keep the animal short of food for some days, because the retained fermenting food might bring on another attack.

**Therapeutics of Acute Tympanites of Sheep.—**

The treatment is nearly the same as that for cattle. We should chiefly rely on pressure upon the left flank and on massage, while the fore-quarters of the animal are kept in a raised position. The use of the oesophageal tube cannot be recommended with sheep, and it is manifest that puncture of the rumen is more dangerous with them than with cattle. We have, therefore, to rely more on internal remedies than when treating large ruminants, and may give half-pint doses of lime water at intervals of 10 to 15 minutes, sulphate of soda ( $\frac{1}{2}$  oz. dissolved in a little water), spirits of camphor (a dessert-spoonful) every quarter of an hour, tincture of colchicum (5 drops), oil of turpentine (half a tablespoonful in  $\frac{1}{4}$  pint of oil), and paraffin oil (half a tablespoonful in the same quantity of spirits). We consider the administration of ammonia to be as hazardous with sheep as it is with cattle. If we have to do with a large number of patients, individual medicinal treatment will generally be out of the question, and we may consequently be compelled to adopt other methods, such as exercising them, or driving them into water in which they can be dipped, or the water poured over them. In desperate cases, puncture of the rumen with the trocar, or, failing it, with the knife, will often be the only remedy left.

*Acute tympanites of pigs* is often observed after the consumption of fermenting food, such as the grains of white beer, which are very rich in yeast; after too copious drinking of warm, sweet whey and after taking poison. As the administration of fluid medicines is accompanied by great danger, the use of the trocar will be the most reliable means of treatment. Veratrum, employed subcutaneously in doses of 1 to 3 gr., may be of benefit.

*Acute tympanites of dogs* occurs in some cases of poisoning (toxic gastritis and enteritis), and in intestinal stenosis.

**CHRONIC TYMPANITES OF RUMINANTS.**

**Nature.**—This chronic tympanites consists in a persistent, obstinate and continually repeated accumulation of gas in the rumen; but not to such a great extent as in acute tympanites. Erdmann found that the composition of the gases in one case was as follows:

Carburetted hydrogen .. ..	..	42 per cent.
Carbonic acid .. ..	..	35 " "
Nitrogen .. ..	..	20 " "

Lungwitz obtained similar results, with traces of S.H<sub>2</sub>. According to him, the gases are chiefly composed of marsh gas and carbonic acid in the proportions of 1·1 to 1·2-1·5, and consequently, there is less carbonic acid in chronic tympanites, than in the acute form.

**Causes.**—Very often chronic tympanites is only a symptom of chronic gastric catarrh, in which case it is brought on by inactivity of the rumen, cessation of rumination and consequent increase of fermentation. We have, therefore, two principal causes of chronic tympanites which are essentially different from each other :—

1. A chronic gastric catarrh, during the presence of which any slight error of diet is apt to give rise to an abnormally large formation of gas.

2. Mechanical impediments to rumination and eructation, such as stenosis of the gullet from new-growths and abscesses. Thus, chronic tympanites, brought on by tubercular degeneration of the lymph glands of the mediastinum, is not infrequently a characteristic symptom of tuberculosis. Sarcomata and actinomycomata of these glands produce a similar effect. New-growths, balls of food, balls of sand and hair balls also prevent the escape of gas. Adhesions of the stomach to the abdominal wall prevent the former from contracting, and thus set up fermentation. These peritoneal adhesions are generally caused by sharp-pointed foreign bodies in the stomach, blows or thrusts on the inferior region of the stomach, and repeated punctures of the paunch (see remarks on traumatic indigestion, page 95). Ruptures of the diaphragm, with prolapse of the reticulum into the thoracic cavity and, possibly, adhesion to the thoracic organs, might greatly interfere with rumination and eructation, and might then produce chronic tympanites. This affection has also been observed as a consequence of tape-worms (*tænia denticulata*).

**Symptoms.**—The symptoms of chronic tympanites are essentially similar to those of chronic gastric catarrh. The principal symptom is a gradually appearing, periodic, intermittent and, finally, obstinate and persistent, though moderate distention of the hollow of the left flank. At the same time rumination is suppressed, the movements and sounds of the rumen are indistinct, and occasionally vomiting is observed. Usually, there is little or no dyspnoea or restlessness. In the

further course of the disease, which often lasts for months, chronic diarrhoea and emaciation may be seen. With respect to prognosis, we may say that tympanites produced by chronic catarrh of the stomach disappears as soon as its cause is removed, but tympanites due to organic changes and displacement of the stomach is incurable. Tuberculosis of the mediastinal glands may be demonstrated by tuberculin.

**Therapeutics.**—Treatment is successful only with the first mentioned form and, in this case, is similar to that of chronic gastric catarrh. We should restrict the fermentation by anti-septics (hydrochloric acid, hyposulphite of soda, creolin and neutral salts), and should stimulate the peristaltic action of the stomach by agents such as tartar emetic, white hellebore, oil of turpentine, etc. Trasbôt has obtained good results from ipecacuanha in doses of 1 to 2 drachms. Eserine cannot be recommended, on account of its unfavourable secondary effects. As a last resource in severe cases, we may make an incision in the left flank, and remove the accumulated fermenting food, and may also employ a permanent canula. The use of the trocar for removing the tympanitic gases is rarely required in the chronic form of the disease. The gas-absorbing remedies have only a temporary influence, and are therefore of no use. Regulation of the diet is, however, of the greatest importance.

#### IMPACTION OF THE RUMEN IN CATTLE.

**Cause.**—This disease, which is frequent among cattle, is generally caused by the too greedy consumption of an unusually large quantity of food that is appreciated by the animal, such as green fodder, new hay, fresh clover, different kinds of brewers' grains and mashes. It also arises from natural greediness, fasting for too long a period and from the drinking of too much water, especially when the fluid is cold.

The symptoms vary according to the quantity, quality, and digestibility of the food that has been eaten. They are usually as follows:—The animal stands back from the feeding trough, arches its back, passes at short intervals and in small quantities normal dung, whisks its tail, kicks with its hind legs against the abdomen, looks round at its hind quarters, while moaning and groaning, occasionally shakes its head violently, and expels air through the nostrils with a hissing sound. Or it stands stupidly and listlessly, without

taking any heed of its surroundings. On rare occasions it may lie down and groan, but get up again after a short time. Appetite is lost, rumination is entirely suppressed, and thirst is sometimes increased. Salivation is often present, in which case the animal goes through the motions of eating without any food in its mouth; occasionally short interrupted eructations escape while the head and neck are stretched out; or peculiar retching movements and true vomiting may take place and be frequently repeated. The muzzle is generally normal. The abdomen, especially on the left side, is greatly increased in circumference. The hollow of the left flank is obliterated and may even project upwards. On palpation of the rumen through the wall of the flank, we find that it is tense, filled up sometimes with doughy, yielding, at other times, very firm chyme, which usually reaches to the upper wall of the rumen, so that percussion gives an empty sound all over this part. Later on, a slight accumulation of gas above the food-contents of the rumen may be found by palpation and percussion. If strong and continued pressure be made on the rumen, the animal will show that it is disagreeable, and try to avoid it. The condition of the paunch can be ascertained by exploration through the rectum, instead of by palpation of the flank. The movements of the rumen, which can be seen and felt, when that organ is in a normal condition, are entirely suppressed, or so much diminished that the hand, when laid on the paunch, can feel no activity in it, and its sounds are greatly diminished or inaudible. There is more or less constipation, and the dung, instead of being passed in flat cakes, is firm, balled and of a dark colour.

The temperature is not increased, and is uniformly distributed; but the pulse is somewhat accelerated and hard. On account of the pressure on the diaphragm, respiration is laborious, and when the animal is recumbent or going down hill, is particularly difficult and even groaning, so that an affection of the lungs might be suspected. The expression is anxious and staring, the eye-balls are prominent, and the conjunctiva is injected.

**Course.**—The intensity of the disease varies. If the rumen be greatly overloaded, symptoms of suffocation and apoplexy may quickly appear, as frequently happens in acute tympanites. In other cases an acute gastro-intestinal catarrh becomes developed. In most cases the attack passes off,

especially if the animal was previously healthy and strong, the quantity eaten not too great, nor the food too indigestible. In mild cases recovery may take place in a few hours, and the signs of digestion usually appear within the first 24 to 36 hours. Rumination and appetite reappear, the movements of the paunch become active, frequent evacuation of gases and copious defecation take place, and recovery is complete in three and four days.

**Differential Diagnosis.**—Overfilling of the rumen may be confused with acute tympanites and, less frequently, with pelvic hernia. The mistake arises in the former case, because acute tympanites appears very rapidly, and directly after feeding, and shows increase in the size of the paunch; and in the latter, because the hernia occurs suddenly, and gives rise to symptoms of uneasiness. An exploration of the rectum guards us from a confusion with ventral hernia; because in impaction of the rumen there is no tense cord in front of the entrance to the pelvis, on its right side towards the abdominal ring. The history of the case is also a good guide. In tympanites the distension of the rumen is greater, and we find by palpation an accumulation of gases and not of firm food. The gas developed in cases of impaction of the stomach, exists only in small quantities and, as a rule, soon passes off; but the disturbances of respiration are of a more severe character. Torsion of the uterus may also simulate impaction of the stomach; because it causes sudden refusal of food, inflation of the left flank, and constipation. Beel and John advise that an examination *per vaginam* should be made in pregnant cows which show symptoms of impaction. An accidental confusion with acute gastro-intestinal catarrh can be easily avoided by paying attention to the history of the case (sudden appearance of severe symptoms). Even the layman seldom fails to recognise impaction of the rumen.

**Treatment.**—In slight cases a purely expectant method of treatment will suffice, and nothing further need be done than to keep the cattle short of food for several days, and to exercise them well. In severer cases we should try to stimulate the rumen externally by massage (pressing, kneading and rubbing from the left flank), and internally by aperients and emetics. Aloes, tartar emetic and sulphate of soda are given in large doses. Here, the best emetic is white hellebore root,

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(150 to 300 grains; or tincture,  $\frac{1}{2}$  oz). Trasbot states that ipecacuanha is of very great value, either at the beginning of the attack, or to complete the effect of puncture. As a last resource, we may make an incision into the rumen and remove a portion of the accumulated food.

## VACUITY OF THE RUMEN.

Under the name of "Vacuity of the Rumen," or "Railway Sickness," Voigtländer has described a peculiar and very obscure morbid condition of cows far advanced in pregnancy, which was produced by rapid and almost entire removal of the contents of the paunch. The animals in question had been carried for a long distance by rail, during which time no food was given, and previous to departure they had only a small quantity of grass, which was digested at an early period of the journey. According to Voigtländer, the symptoms closely resembled those of paralytic milk fever. The cows, being completely paralysed, were unable to stand; the head was turned to one side; the eyes were deep-sunken and dim; the pupils dilated and respiration greatly accelerated. Only a few recovered. On *post-mortem* examination, the rumen was found to be so empty, that it could be wrapped round the arm like an empty bag. Viegel found the same symptoms in cows that had suffered from similar conditions. The cows, especially those that were in good condition, became ill soon after their arrival, and died in from eight to twenty-four hours. The most prominent symptoms were: High fever, complete loss of appetite, continued lying down and a paralytic condition of the organs of digestion. *Post-mortem* examination showed that the rumen was inflated and nearly empty; the liver hyperæmic or clay-coloured, the small intestines very red; and peritonitis and hyperæmia of the uterus were sometimes present. Weigel ascribes the disease to long-continued empty condition of the paunch (for two or three days), want of rest from not being allowed to lie down, and spinal concussion. Röder reports similar cases. The observations which have been made up to the present do not give us much exact information as to the nature of this disease.

## VOMITING.

Vomiting occurs in impaction of the rumen, in some affections of the oesophagus (*diverticula*), in invagination (intussusception) of the intestine, and from the use of eserine, eseridine, pilocarpine, arecoline, veratrine and apomorphine. In other cases the causes of vomiting are unknown.

CATARRH OF THE ABOMASUM AND SMALL INTESTINE  
IN CATTLE.

As already stated, the four compartments of the stomach of ruminants may be respectively attacked by a primary catarrhal inflammation. Thus it seems that the abomasum is not uncommonly the seat of a primary affection, the correct diagnosis of which is generally very difficult. Prietsch found among 600 apparently healthy cattle which were slaughtered, from 20 to 25 per cent. that had either acute or chronic catarrh of the abomasum. In the majority of slaughtered cattle Ostertag found under the epithelium of the mucous membrane of the abomasum a parasite, the *Strongylus contortus*, which he regards as the cause of emaciation and hydramic cachexia, when it invades that organ in large numbers. As descriptions of catarrh of the abomasum are contradictory in some essential points, we shall content ourselves with briefly referring to observations which are least in dispute.

Saake has met with catarrh of the abomasum in young cattle, oxen and cows before and after calving. In the beginning the symptoms are the same as in other gastric affections. During a series of alternate improvements and relapses gas becomes frequently developed in the rumen, although the animals may look remarkably well, in spite of considerable tympanites. On auscultation of the rumen we may hear a peculiar and frequently loud metallic sound, which Saake considers to be a characteristic symptom of catarrh of the abomasum, as it is never heard in a case of ordinary inflation. Later on, severe diarrhoea sets in, with gradual emaciation followed by a chlorotic condition, and a tendency to oedema in the intermaxillary space. The appetite becomes vitiated and capricious, without well-marked pica being present; and the right hypochondrium becomes sensitive to pressure. The course of the disease is very long, and often ends in death after several months. On *post-mortem* examination, we find considerable swelling of the mucosa and submucosa, which are oedematosly infiltrated and sometimes 1½ inches thick at the pylorus and beginning of the duodenum; and catarrhal reddening, especially in the pylorus, and in chronic cases follicular ulcers of the mucous membrane. Tannin in doses up to 1½ oz. has proved very effective in this disease. Under the name of "Catarrh of the Abomasum and Small Intestine," Harms has described a suddenly appearing affection of the organs of digestion, which takes either an acute or a chronic course, and is distinguished from ordinary gastro-intestinal catarrh by icteric colouring of the conjunctiva, normal

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condition of the feces, unimpaired activity of the rumen (even when it is filled), and manifestations of pain, on pressure being applied to the region of the abomasum. Later on symptoms of pica make their appearance. On *post-mortem* examination of the early stages, we find oedematous swelling, hyperæmia and a mucous coating; and in those of older standing the mucous membrane is thickened and has abnormally large folds. As regards treatment, Harms recommends low diet, easily digestible food and the administration of Carlsbad salt and hydrochloric acid. He has described a case of impaction of the abomasum, which contained 78 pints of ruminated food and, consequently, if viewed from a distance, might have been mistaken for the rumen in an inflated condition. The symptoms during life were frequent eructations, pain on pressure being applied to the region of the abomasum, salivation, vomiting and great obstruction of the rectum by feces. Kitt has described a case of scirrhus of the abomasum.

### GASTRO-INTESTINAL CATARRH OF YOUNG CATTLE.

Gastro-intestinal catarrh of young cattle (sucklings and newly-weaned calves) deserves to be discussed separately, as it is a subject of great importance to breeders of cattle and graziers, and is essentially different from the same disease in grown-up cattle, both as regards its causes, course and treatment. Also, it is frequently confounded with infectious dysentery ("White Scour").

**Causes of the Disease in Sucklings.**—I. Diseases and abnormal conditions in the mother. The physical state of the cow is closely connected with the health of the suckling by means of the milk, particularly with regard to digestive diseases, mal-nutrition, cachexia and debility. Infective diseases, such as foot-and-mouth, general tuberculosis, and tuberculosis of the udder, are most dangerous. Mastitis and even temporary inflammatory swelling of the udder very often cause gastro-intestinal catarrh in the calf. The feeding of the cow is also of the greatest importance. Food which is too fattening, too concentrated and too watery, or food which is decomposing and otherwise unwholesome, may produce indigestion in the calf, in consequence of their bad effect on the milk. In this respect the chief lacteal faults are—too much or too little fat in the milk; too little or too much casein; tendency of the milk to coagulate in the udder; and a bitter, rancid or ropy condition of the milk. Foreign sub-

stances (such as ethereal oils, resinous and pungent matters, poisons and medicines, especially aperient salts) in the milk may exercise an irritating effect on the mucous membrane of the calf's stomach. Over-exertion on the part of the cow may also affect the health of her young one, by impairing the quality, or diminishing the quantity of the milk. Some authorities consider that the usual practice of removing the colostrum by milking is a cause of this disease.

2. Errors of diet during lactation. Here the most frequent cause is the consumption of too large a quantity of milk at one sucking, after the calf has been separated for a comparatively long time from the cow, and has consequently become unusually hungry. The giving of milk-substitutes may also bring on catarrh.

3. Chills received by the newly-born calf, from a cold and damp stall, and from a cold udder.

4. Retention of the meconium, in which case the resulting constipation leads to decomposition of the intestinal contents and irritation of its mucous membrane by the products of decomposition.

#### **Causes of the Disease in Newly-Weaned Calves.—**

1. Faulty weaning, such as too early feeding on dry food, especially coarse, fibrous and irritating fodder, which can hardly fail to produce a morbid condition of the stomach and intestines ; mouldy forage and straw which has been cut too short.

2. Improper milk-substitutes, particularly those which are too rich in starch. They set up inflammation of the intestinal mucous membrane by the production of acetous, lactic and butyric fermentations.

3. Bad weather, especially during the spring and autumn ; and drinking too cold water.

4. Shedding of the milk-teeth, during which time the calves gnaw and swallow all kinds of objects.

5. The presence in the alimentary canal of worms (as for instance, *strongylus contortus* in stomach-worm plague of lambs ; *taenia expansa* in tapeworm diseases of lambs ; and *strongylus armatus* in sucking foals). Some animals are predisposed to gastro-intestinal catarrh from congenital weakness of digestion, diseases of the mesenteric glands, etc. Every newly-born animal is liable to it, in consequence of the great sensitiveness of its organs of digestion.

**Anatomy.**—On the whole, the anatomical changes in the stomach and intestine are the same as those met with in adults, except that in fatal cases they are not always in proportion to the excessively severe symptoms exhibited during life. The contents of the stomach of sucklings usually consist of firm, lumpy, caseous clots; and those of newly-weaned animals, of balls of coarse hay and straw. In sucklings the normal coagulated condition of the stomach is not uncommonly regarded as abnormal. The gastro-intestinal mucous membrane shows various catarrhal changes, such as congestion, swelling, follicular catarrh, and even ulcers in cases of acute catarrh; and atrophy, thickening, prominences, pigmentation, etc., in the chronic form. The mesenteric glands are generally more or less swollen. In many cases the carcasses are greatly emaciated and show signs of anaemia and hydremia.

**Symptoms.**—The symptoms may appear either gradually or suddenly, according to the cause. In sucklings, diarrhoea, which is the principal symptom, may be preceded only by decreased inclination to suck, slight debility and depression. If the disease be due to errors of diet or to chill, the symptoms, especially in newly-weaned animals, will appear suddenly in the form of fever, well-marked constitutional disturbance, unequally distributed temperature, cold legs, dry muzzle, debility and depression. The diarrhoea, which soon sets in, consists at first of thick pasty faeces of normal colour and smell; later on, it becomes watery and light coloured, and is frequently expelled in jets; and, finally, it is greasy, yellow or dull-green, frothy, sour-smelling, or very stinking and intermixed with flakes and lumps. Towards the end the dung shows streaks of blood, or may assume a sanguineous colour; is passed involuntarily; soils the hind legs and tail; and by its corrosive action, frequently causes the hair in the neighbourhood of the anus to fall out. Tenesmus and symptoms of slight colic are present; the animal stands with its back arched and the hind legs placed under the body; the skin is dry; the hair stands on end; the effluvium from the body is very foetid, and the animal becomes excessively faint and weak. In combination with the diarrhoea we often observe the repeated appearance of tympany of the paunch and intestines. This inflation is generated by abnormal decompositions which occur during the course of the gastro-intestinal catarrh, and may become developed before, after, or simultaneously with the diarrhoea.

The course of the disease may be either acute or chronic. Death may occur in a few days, or may be deferred for several weeks. In most cases death is due to exhaustion, general anaemia and hydramia. The disease is sometimes complicated with catarrhal pneumonia, which is probably a result of atelectasis (imperfect expansion), as may frequently occur in weak animals immediately after birth. Simple intestinal catarrh can be distinguished from dysentery by its more sporadic occurrence, its milder character, its more favourable course, and the fact that it does not appear so soon after birth as infective dysentery.

**Prognosis.**—In this disease prognosis should always be made with caution, especially if the diarrhoea and tympanites are of long duration.

**Treatment.**—In this respect diet occupies the first and medicine the second position. With the object of removing the cause in a suckling, treatment should be chiefly applied to the mother. All factors which unfavourably influence the quality of the cow's milk, especially too fattening food, bad maintenance, and too long retention of the milk in the udder, should be removed. If the illness of the calf can with certainty be traced to the cow, the former animal should be withdrawn from the pernicious influence of the latter, by putting it to another cow which is healthy. If the diarrhoea is due to improper feeding, the error should be corrected without delay. With sucklings one must take care that they do not consume too much milk at a time, little and often being the best rule with them, because their stomachs are not suited for the digestion of large quantities. If we cannot allow the calves to suck when they like, we should give them an opportunity of doing so at least three or four times daily. With calves about to be weaned, we must not wean them too suddenly. For instance, calves should not be weaned before the age of four to six weeks, and lambs three to four months. In both cases the process should be carried out gradually, and all stimulating and indigestible food avoided.

To combat the diarrhoea we may at first use slightly astringent and mucilaginous domestic medicines, such as infusions of toast, parched meal, coffee, marshmallow, gum, hemp-seed, poppy-seed, almonds and glue, raw eggs and gruel. As regards medicines, we may begin with a slight aperient, especially if fermenting substances have accumulated in the intes-

tines. For instance, to calves we may give two or three tablespoonfuls of castor oil or linseed oil; or 1 oz. of Glauber's or Epsom salts, in a mucilaginous infusion (quarter of the dose for lambs). Soluble cream of tartar has been recommended as a laxative (calves  $\frac{1}{2}$  to 1 oz., and lambs 75 to 150 grains).

For abnormal fermentations in the intestinal canal, we may give creolin (calves 1 dr., and lambs 10 to 15 min.) in cold water two or three times a day. Also, carbonate of magnesia, carbonate of soda, chalk, bone meal, creosote (calves 3 to 5 drops, lambs  $\frac{1}{2}$  to 1 drop), tar (in the form of tar-water by the mouth, or as an enema), salicylic acid (calves 15 to 30 grains, lambs 4 to 8 grains in spirit) and resorcin.

In severe cases of diarrhoea, opium (powder, tincture, or as Dover's powder) and rhubarb render good service. We may try enemas of a one per cent. solution of alum or tannin.

#### TRAUMATIC INDIGESTION IN CATTLE.

**General Remarks.**—The literature on the subject of disturbances caused by foreign bodies in the stomach of ruminants is very rich. In most cases it refers to so-called traumatic carditis and traumatic pericarditis, which are complications or stages of the purely gastric affection that we are at present discussing. Traumatic disease of the heart will be dealt with later on (p. 364). Foreign bodies are found very frequently in the stomach of ruminants, especially in cows, and without any previous manifestation of disease. It has been reported that a carving knife remained three months in the stomach of a beast without causing any injury. In other cases, foreign bodies produce more or less grave lesions in the reticulum and adjoining organs, particularly in the diaphragm. The intensity of the affection depends, above all things, on the external condition of the foreign bodies. The following is an appropriate classification :—1. Blunt bodies, as, for instance, stones. 2. Pointed bodies, which, like nails that have a head, penetrate the wall of the stomach and remain fixed in it. 3. Pointed bodies which pass completely through the wall of the stomach, as, for example, needles and pieces of wire.

**Etiology.**—There are several reasons why foreign bodies are found more frequently in the stomachs of ruminants, and especially in those of cattle, than in other domestic animals. First of all, cattle have the habit of licking objects within reach and swallowing them if possible. They consume food very rapidly and chew it imperfectly in the first instance, leaving its comminution to be completed during rumination. Cows with

female attendants, who are, we might say, covered with hairpins and needles, are more liable to suffer from traumatic inflammation of the stomach than cows which are tended by men. The disease is met with more rarely on large farms than on small holdings where kitchen refuse of every kind is used for cattle-food. The inner wall of the stomach of cows kept by shoemakers is sometimes almost paved with nails. The disease is also comparatively common in the neighbourhood of stocking and needle factories and in byres with shingle roofs. It is more frequent in stalls than on pasture. Harms found that animals which stand at the lower end of the feeding trough are more apt to swallow foreign bodies, which readily accumulate at the end of the slightly sloping trough.

**Anatomy.**—The *blunt* foreign bodies which are found in the reticulum are usually stones or sand; but we may also find balls of hair, articles of clothing, pieces of leather, straps, broken handles of whips, balls, spoons, coins and even snakes. These bodies produce catarrhal or inflammatory processes in the mucous membrane of the stomach. In impaction of the stomach with sand, the grains of silica form a hard, compact mass, and the mucous membrane is densely incrusted with sand.

The *pointed* foreign bodies which do not pass completely through the wall of the stomach are usually nails with a head, pins, hairpins and bent pieces of wire. They are generally found in the reticulum because the honeycombed structure of its surface is peculiarly well adapted to arrest the progress of these bodies. At the place penetrated by the foreign matter, the wall of the reticulum shows a circumscribed inflammation which conceals a narrow, fistulous canal that has an indurated wall of a grey or dull-blue colour. This canal contains pus mixed with particles of food. Around this lesion the peritoneal surface of the reticulum is often adherent to adjacent organs (particularly to the diaphragm, which is also inflamed), by means of a plastic exudation, often half-an inch thick. Abscesses may be found near the adhesion; and, less frequently, the diaphragm may show deposits of granulation tissue, which may be as much as an inch in thickness. The rumen is often adherent to the abdominal wall, and is generally filled with food. Perforating foreign bodies are very seldom met with in the omasum and abomasum. The foreign matter often quits the wound in the reticulum and re-enters that compartment. In many cases it cannot be found, on account of its having been evacuated along

with the dung, a fact which formerly gave rise to the erroneous supposition that the foreign body had been dissolved.

Pointed bodies which pass completely through the wall of the stomach are generally needles of all kinds, pieces of wire, broken nails, pointed pieces of iron, knives, scissors, forks, broken bits of glass, splinters of wood, broken particles of stone, broken sticks, umbrella ribs, etc. Besides the above-described lesions of the reticulum and diaphragm, we may observe the following changes, according to the direction the foreign body has taken : extensive peritonitis, abscesses of the liver, spleen and lungs, adhesions of the reticulum to the liver, abscesses in the thoracic and abdominal walls, lobular pneumonia, adhesions of the lungs to the diaphragm, pleuritis, etc. Sometimes the foreign body may penetrate into the spinal cord, ribs, muscles of the thigh, etc. Its passage is marked by abscesses, inflammation, formation of fistula and adhesions. In rare cases death by haemorrhage into the reticulum and rumen has been observed. The changes which occur in the pericardium and substance of the heart are described under the heading of "Traumatic Carditis."

**Symptoms.**—The symptoms caused by the presence of blunt bodies in the antestomachs of cattle are, on the whole, chronic or acute disturbances of digestion, which are due to irritation, contusion and superficial injury of the mucous membrane. Graver symptoms are found after devouring considerable quantities of sand, such as (according to Krichels-Schmidt) continuous chewing without swallowing, slavering, groaning, constipation, frequent lying down, arching of the back and gradually increasing weakness. We find on palpation that the rumen is as hard as a stone, and that the peristalsis of the stomach is diminished and finally ceases. In fatal cases, death occurs in from nine to seventeen days. Chronic indigestion is a sequel in some cases.

When the foreign body is pointed, the symptoms are generally those of inflammation of the stomach and diaphragm, and are usually as follows :—Without any apparent cause we find disturbances of digestion with suddenly developed symptoms which resemble those of invagination of the intestines, and which consist in attacks of colic, tottering hither and thither, kicking at the abdomen, etc. In other cases, only the symptoms of acute catarrh of the stomach appear at the beginning of the seizure ; but subsequently a number of chronic, periodically recurring

gastric attacks of greatly varying character, among which chronic tympanites is the most prominent. The fact that this symptom does not yield to any ordinary treatment is of the greatest diagnostic value. If the affection last for a long time, it gradually brings on great emaciation and debility. The following factors are of great diagnostic weight: The animals manifest intense pain on various occasions by groaning, as, for instance, after feeding, when lying down, getting up, walking about, and particularly during movement, which is careful and stiff, and the expression of the face shows anxiety and pain. Usually palpation of the reticulum is very painful. We may note that the place for this palpation is on the left side of the ensiform cartilage, on the inferior abdominal wall. Frequently the animal also evinces pain on pressure of the sternum, thorax, withers and paunch. On percussion being applied to the diaphragm at its points of insertion, pain is manifested; and in the same way we sometimes find a circumscribed area of dulness in front of the diaphragm (abscesses, granulating new growths, etc.). The contractions of the diaphragm during inspiration and defecation and its movements during expiration are painful and accompanied by groaning. Hence, abdominal pressure for purposes of defecation is used as little as possible, and consequently an accumulation of faeces forms in the rectum. If a large vessel be injured by a foreign body, we may have " discharge of blood from the rectum.

If the pointed body pass completely through the stomach and diaphragm, we shall have other symptoms caused by its migratory tendency, which latter is due to the contractions of the reticulum and to the movements of the diaphragm, especially during expiration, by which the pointed body is moved towards the thoracic cavity. In other instances it reaches the abdominal cavity by means of the peristalsis of the stomach. Strained abdominal pressure during calving, and highly-wrought action of the diaphragm during forced breathing, as in transport to and from market, usually accelerate the forward propulsion of the foreign body. According as various organs are injured by the foreign body, the symptoms of traumatic inflammation of the stomach and diaphragm become complicated with those of carditis, pneumonia, pleuritis, pneumo-thorax, enteritis, peritonitis, hepatitis, pyæmia, septicæmia and fatal internal haemorrhage. With respect to these complications and the further course of the disease, readers may consult the remarks on traumatic pericarditis. If the foreign body finally work its

way to the surface, we shall find a swelling on the affected part of the skin which will be very characteristic of this stage of the affection. This swelling is generally found on the left wall of the thorax, in the region of the left inferior ribs (infra-costal region), region of the sternum, or behind the elbow. It may appear on the right side of the thorax, and even on the abdomen, navel, flanks or udder. This phlegmonous swelling varies in size up to that of a man's head; is, as a rule, only moderately painful; of varying consistence; and eventually forms an abscess which contains ichorous pus mixed with particles of food, sand and even, perhaps, the foreign body itself. These substances may be discharged by means of a fistula, through which we may sometimes pass a probe into the stomach. This fistula often does not close for several weeks, or even months, and recovery may not take place until after a long period of ill-health.

**Differential Diagnosis.**—Diagnosis, which is of varying difficulty, can generally be made only by an exact examination, when the more important symptoms are present.

Confusions chiefly occur with chronic simple catarrh of the stomach, in which the manifestation of pain on palpation of the affected organs is absent; the gastric disturbances are not of such a varying nature; apparent causes are generally to be found; proper treatment leads to recovery, or at least to improvement; and emaciation is not of such a rapid and intense type.

The separation of traumatic inflammation of the stomach and diaphragm from peritonitis and gastro-enteritis may be difficult, although the two last-mentioned diseases are usually distinguished by a more rapid course, higher fever and graver disturbance of the general health. If the foreign body have penetrated into the lungs, it may produce symptoms of a chronic pulmonary affection which cannot always be distinguished from tuberculosis of the lungs, unless a previous indigestion, a demonstrable affection of the diaphragm, or the absence of the extensive bronchial catarrh which usually accompanies tuberculosis of the lungs, indicate the presence of a foreign body. The disease we are considering has also been confounded with pleuro-pneumonia, which is epizootic in its occurrence, typical in its course, and whose symptoms are essentially pneumonic. It also presents no disturbances of the stomach and diaphragm.

**Treatment.**—With regard to prophylaxis, we should replace female attendants by male ones, or else properly instruct them, and frequently examine the lower strata of the food. Harms proposes to fix at each end of the feeding trough a receptacle for foreign bodies. It is most important to make an early diagnosis. So long as the diagnosis cannot be made with certainty, the treatment should be the same as that for acute or chronic catarrh of the stomach. If we are certain that the cause of the disease is a foreign body, we shall have to decide between early slaughter or removal by an incision into the rumen; the former means being preferable in all severe cases. Obich's operation should be tried, if the disease be not too far advanced, although some state that they have obtained good results even after pericarditis had set in. Obich's method consists in opening the rumen at the hollow of the left flank, about four inches below the transverse processes of the lumbar vertebræ, by means of a vertical incision, through which the hand can be passed into the rumen forwards and downwards, in searching for the opening of the reticulum, which is on the right hand side, and in feeling on the floor of the reticulum for foreign bodies. After removing these the wounds of the rumen and abdominal wall should be closed; the former by an intestinal suture, the latter by an ordinary suture. Usually the operation is borne very well, and may be performed even during pregnancy. It is not always successful, because severe affection of the heart and diaphragm may be present, and an exact diagnosis is frequently impossible. Out of thirteen operations, Obich had only four good results. It might be tried in cases in which the diagnosis was certain and the disease not too far advanced, unless slaughter was to be preferred for economic reasons. In place of this operation, Schöberl recommends that the beast be placed on its back, and the region on the left side and behind the ensiform cartilage be energetically kneaded with the foot. He states that out of sixty beasts which were treated in this way, the foreign body failed only on two occasions to return into the reticulum.

#### GASTRO-INTESTINAL CATARRH OF CARNIVORA.

**Etiology.**—Impaction of the stomach, catarrh of the stomach, and catarrh of the intestines are far more frequent in dogs than in horses and cattle. In the dog these maladies form about 60 per cent. of all the diseases of digestion. Among

70,000 sick dogs treated at the Berlin Canine Hospital during nine years, there were 4,000 cases (6 per cent.) of gastro-intestinal catarrh. This frequency is probably due to the careless manner in which dogs are usually fed. The common and irrational practice of giving dogs only one meal a day leads to a quotidian overcharging of the stomach. Hounds and bitches in milk suffer from this cause more than other dogs, on account of their greater voracity. Hungry dogs often get impaction of the stomach from eating large quantities of potatoes, bread, bones, etc. Further causes are the consumption of food which is difficult of digestion (like some kinds of dog biscuits), mechanically irritating, spoiled and decomposing food; swallowing foreign bodies (see remarks on foreign bodies in the intestinal canal, p. 108), especially by young, playful dogs, and those which fetch and carry; imperfect teeth; too rapid eating; eating too hot food, and chill, as, for instance, when they are clipped or sent into the water.

Acute gastro-intestinal catarrh in carnivora is an accessory symptom, especially in distemper; and the chronic form of this catarrh is an accessory symptom in several chronic morbid conditions, such as ulcers in the stomach, intestinal worms, Bright's disease, and stasis of the portal vein after chronic affections of the heart, lungs and liver.

**Symptoms.**—1. *Overloading (surfeit) of the stomach* generally produces vomiting, which soon puts things right. If vomiting does not come on, the animal will retch, slaver and turn away with nausea from any food placed before him. Thirst, as a rule, will be increased; pressure on the overfilled stomach will cause the animal to groan with pain; respiration is often greatly accelerated. The patients are very dull, depressed and peevish, or they are uneasy, frequently change their resting-places, groan, whine and show signs of colic. There is no fever.

2. *Gastric catarrh.* If a distinction between catarrh of the stomach and catarrh of the intestines be possible in our domestic animals, it can best be made in carnivora, because in the former the most important symptom, vomiting, is seldom absent. We must, however, bear in mind that it is not a characteristic symptom of gastric catarrh, because it also occurs in various other morbid conditions.

The course of gastric catarrh in carnivora may be acute or chronic, and with or without fever. In mild cases there is nothing morbid to be seen, except dislike to exercise, depression

of spirits, decreased or capricious appetite and increased thirst, which may all pass away in a day or two. Usually retching and vomiting make their appearance. At first, the vomited matter consists of normal chyme ; but later on, of ropy, glairy mucus, which contains air-bubbles, and sometimes bloody or yellow streaks ; and finally of more or less pure bile, especially in cases of severe vomiting. As a rule, there is great thirst, and the consequent increased consumption of water gives rise to renewed vomiting. There is little or no appetite, and any food which is taken is generally returned. There is more or less constipation. The temperature of the body is unequally distributed, the nose warm, often dry, the rate of the pulse accelerated (by 20 or more beats), and the temperature raised (sometimes to  $103^{\circ}$  F.). The animal is depressed, lies down a good deal, avoids his usual place of rest, scratches away the litter and prefers the cold floor. Pressure on the stomach is often painful.

3. *Intestinal catarrh.* If the intestine be the chief seat of the disease, vomiting will generally be absent ; the appetite will be more or less impaired, and the stomach will not be abnormally sensitive to pressure. The most noteworthy symptom is diarrhoea, which is always present, owing to the shortness of the intestinal canal of carnivora ; and it varies a good deal in severity. In mild cases the faeces have a pappy consistence, but in severe attacks they are watery or mucous (slimy), sometimes yeast-like, bilious, tinged with blood, occasionally very frothy, and usually foetid ; they often contain particles of undigested food, and they soil the parts about the anus. Tenesmus makes its appearance with frequent protrusion of the congested and swollen mucous membrane of the rectum. Prolapse of the rectum may occur in young and weak animals. Biliary pigments are constantly found in the urine. Fever is seldom absent in intestinal catarrh, and the temperature may rise to  $106^{\circ}$  F. Symptoms of jaundice, owing to the severity of the disease in the duodenum, is more often seen in dogs and cats than in horses and cattle.

**Prognosis.**—Acute gastro-intestinal catarrh in carnivora generally ends in recovery. Only very young and debilitated animals, especially if suffering from severe diarrhoea, become greatly exhausted, and even die. Icteric symptoms render prognosis unfavourable. The fact that biliary pigments are frequently found in the urine shows that chronic catarrh, probably arising from errors of feeding, is more common in

these animals than is usually supposed, and its prognosis is less favourable than that of the acute form. The chronic cases, in which diarrhoea persists for weeks and months, are generally incurable, because they usually have their origin in lesions of the intestinal mucous membrane, as, for instance, atrophy of the epithelium with hypertrophy of the connective tissue of the mucous membrane, tubercular deposits and new growths.

**Treatment.**—Purely dietetic treatment suffices in many cases and may consist in depriving the patient of food and drink for some time, and we should above all things try to prevent the immoderate consumption of water. We have obtained a cure in many cases, merely by a change of food, especially when the animal has been fed on dog-biscuits, which are frequently injurious. The best food is raw meat given often and in small quantities. In cases of impaction of the stomach and in those of gastric catarrh caused by the presence of hurtful food in the stomach, it is best to empty the stomach as soon as possible by an emetic. This procedure cures the gastric catarrh and prevents the occurrence of intestinal catarrh, which might otherwise follow as a consequence of the further advance of the irritating ingesta. In selecting an emetic, we should choose one which is least liable to irritate the stomach, as, for instance, hydrochlorate of apomorphine subcutaneously. Ipecacuanha and tartar emetic may also be used.

If there be loss of appetite, we may give hydrochloric acid (a teaspoonful to a tablespoonful of a mixture of 85 minimis in 8 oz. of water), with or without an equal quantity of pepsin; tincture of gentian with hydrochloric acid, pepsin wine, tincture of cinchona and condurango wine.

For diarrhoea we may at first prescribe a diet of dry food, toast water, rice water, barley water, and hydropathic packing of the abdomen; or we may give a teaspoonful to a tablespoonful of port wine. Later on, we may use laudanum or Dover's powder and, in very obstinate cases, nitrate of silver ( $\frac{1}{15}$  to  $\frac{1}{2}$  grain in a pill). Prior to this, we may employ enemas of solutions of alum, tannin or nitrate of silver (from 1 to 2 per cent.), which we have found to be very efficacious in some cases.

For constipation, which is seldom persistent in this disease, we may give calomel ( $\frac{1}{2}$  to  $1\frac{1}{2}$  grains), which not only acts as a purgative, but also disinfects the intestinal canal. We are

only rarely obliged to combat vomiting in cases of gastro-intestinal catarrh of carnivora. If it become troublesome, we may in the first instance give small pieces of ice, or ice-cream in the case of delicate dogs; or we may give small doses of opium ( $\frac{1}{2}$  to 3 grains), tannin ( $\frac{1}{2}$  to 7 grains), bi-carbonate of soda (7 to 15 grains), bromide of potassium (15 to 50 grains per rectum), oxynitrate of bismuth, creasote, creolin, chloretone, hydrocyanic acid and tincture of iodine. For abnormal fermentation, we may give calomel, creolin (17 to 35 minims), and naphthaline (8 to 16 minims). After recovery, the mucous membrane of the stomach and intestines should be protected for some time from irritation by the avoidance of food which is difficult to digest.

#### CONSTIPATION IN DOGS.

**Etiology.**—Constipation occurs more frequently in dogs than in other domestic animals. Out of 70,000 sick dogs that were treated at the Berlin Canine Hospital during nine years, there were 1,275 (2 per cent.) cases of constipation. The disease, as a rule, takes the form of insufficient peristaltic action, or mechanical obstruction. The chief causes are as follows:—

1. Food which is difficult of digestion or which is non-stimulating, especially too abundant feeding on bones; exclusive feeding on bread, dog-biscuits, pulse, porridge and dry food.
2. Insufficient exercise, particularly with dogs that are kept in-doors or chained up.
3. Old age, in which peristalsis becomes diminished, on account of atrophy of the intestinal mucous membrane and of the muscular coat of the intestines.
4. An existing chronic intestinal catarrh, which diminishes the sensitiveness of the mucous membrane by setting up atrophy in it, and by covering it with mucus, and which also decreases secretion of the intestinal juices, and reduces the activity of the reflex movements of the intestines.
5. Mechanical obstacles which impede the passage of the intestinal contents, as we find in constipation brought on by foreign bodies and accumulations of dung (*koprostasis*), especially dung which is full of bones; stenosis of the intestine by compressing tumours, among which, swelling of the prostate gland plays a prominent part; pressure on the intestine by dis-

charged abscesses ; dilatations and paralysis of the intestine and diverticula of the rectum ; piles ; engorgement of the anal glands, and obstruction from outside by sticky and felted hair (so-called " false constipation ").

6. Constipation is also a *symptom* of various febrile diseases, in which the secretion of the intestinal juices becomes diminished ; of all chronic debilitating maladies, which bring on relaxation and mal-nutrition of the intestinal wall ; of acute gastro-intestinal catarrh after previous diarrhoea and after the too frequent administration of laxatives ; of jaundice caused by absence of bile, which should stimulate the peristaltic movements of the intestines ; of peritonitis and inflammation of the intestine due to oedematous infiltration and paralysis of the intestinal wall ; of spinal paralysis and cerebral disease, in which the constipation is a manifestation of disturbed intestinal innervation ; of diseases of the diaphragm and intestinal wall in consequence of diminished activity of abdominal pressure ; of muscular rheumatism, especially of lumbago ; of increased lactation, etc.

**Symptoms.**—Dogs are less liable to constipation from colic than horses. The chief symptom is the more or less complete suppression of defecation. The patient makes frequent, violent, and occasionally painful efforts to evacuate, and is either unsuccessful, or passes small, dry, hard, single balls of excrement, which often contain undigested pieces of bones that are usually in an earthy and stinking condition and sometimes covered with mucus and blood. The appetite is at first decreased and, later on, is suppressed for days and even weeks. Peristalsis is also in abeyance, and the abdomen is sometimes tense and tucked up ; at other times, tympanitic. On palpation of the posterior part of the body, which may be rendered very difficult on account of the tension of the abdominal wall, we usually find, at the entrance into the pelvic cavity, a hard, nodular or sausage-shaped mass of faeces (a so-called " faecal tumour "). This faecal cord may reach as far forward as the ensiform cartilage, and its palpation generally causes pain. The parts around the anus are often inflamed, swollen and soiled with yellowish, gritty excrement. In cases of so-called " false constipation," the tenacious, semi-solid stools form, with the hair around the anus, a stiff mass that renders defecation impossible. On exploration of the rectum, which is usually very painful, we find the temperature raised, the

mucous membrane dry and swollen, and the rectum often filled with excrement chiefly composed of bony particles. In other cases, we fail to reach the accumulated faecal masses, or succeed only in touching them with the end of the finger. Sometimes the excrement which had collected in front of the "faecal tumour," burrows its way through it, and may appear in the form of stinking diarrhoea intermixed with a good deal of gas. Vomiting often occurs in cases of complete intestinal obstruction.

Fever is either entirely absent, or of a very mild type in cases of long-continued constipation. Depression gradually increases; colic occasionally appears, and exercise is avoided. Male dogs now and then micturate without raising a hind leg. The peculiar manner in which the tail is stretched out, or considerably bent at the root, is very characteristic, and is often sufficient for diagnosis.

**Course.**—Generally speaking, the disease lasts from a few days to a month or six weeks. In more prolonged cases, the action of the accumulated faeces on the intestinal mucous membrane gives rise to symptoms of diphtheritic intestinal inflammation, intestinal necrosis, septicaemia and perforating peritonitis. Cicatricial strictures of the mucous membrane and diverticulae of the intestinal canal are sequelæ which may cause recurrent attacks of constipation. The average duration is from a week to a fortnight, and the disease generally ends in recovery. With regard to differential diagnosis, it may happen that failure to pass faeces, as a consequence of long fasting, may be mistaken for constipation. Confusion between "faecal tumour" and true new-growths may also occur, although the differentiation will be easy, if we pay attention to the form, hardness and position of the swelling, to the history of the case and to the further course of the disease. In difficult cases, we may be able to demonstrate then on-existence of a new-growth by the successful employment of aperients and enemas.

**Treatment.**—Treatment is usually dietetic, mechanical and medicinal. In very rare cases, such as those of abscesses, swelling of the prostate gland and foreign bodies in the intestine, surgical aid may be required.

As regards *diet*, bulky and indigestible food (bread, bones, dog-biscuits, potatoes, etc.), should be avoided. At first, we

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may keep the patient fasting, and allow him only fresh cold water. In long standing cases, we should feed him only on milk, salt meat broth and, possibly, lean meat free from bones. At the same time, we should try to stimulate peristalsis by frequent exercise.

Among *mechanical* means, ordinary enemas or injections of large quantities of water by means of an india-rubber pump are very effective. Pure warm water is best for softening and washing out the accumulated faeces. Clysters of cold water are to be preferred only in cases of intestinal paresis. We may try injections of from 7 to 14 oz. of olive oil, three times daily. Glycerine is generally useless for this purpose. We should also try to remove the constipation by frequent massage, namely, by carefully pressing and kneading the obstructing faecal ball from the outside, and by exploring the rectum with the finger, forceps or curette. These manipulations demand the expenditure of much time, patience and trouble.

These mechanical means may be supplemented by the use of purgatives, such as castor-oil or calomel. In very obstinate cases, supposing that the intestine is in no way inflamed, we may try from 1 to 5 drops of croton oil, with gum arabic and castor oil. We should bear in mind that the strongest cathartics will be unable to remove the ball of faeces, if it be wedged in the intestinal canal, or if the intestinal wall be paralysed. After recovery, the dog should be appropriately fed, so as to avoid a relapse.

### CONSTIPATION IN CATS.

Constipation is rare in cats and, when it occurs, is generally caused by foreign bodies, diverticula and new-growths. Von Puntigam has described a case of feline constipation caused by a recto-vaginal fistula. The treatment consists of enemas and the administration of calomel.

## FOREIGN BODIES SWALLOWED BY DOGS.

**Etiology.**—As in cattle, foreign bodies cause in dogs, and less frequently in cats, peculiar morbid conditions which may not be diagnosed during life, and are often mistaken for symptoms of rabies. In some cases these foreign bodies cause haemorrhages and perforation of the stomach and intestine, with fistulae, which accidents are purely surgical. Other

affections produced by these bodies will be discussed later on under the heading of "Perforative Peritonitis." A third variety of these accidents gives rise to dangerous gastric symptoms by impaction of the stomach or intestines, or by a more or less complete obstruction of the intestinal lumen, and by severe haemorrhagic and necrotic inflammation of the mucous membrane.

Foreign bodies, which cause the accidents in question, are usually large pieces of bone, stones, metal and glass balls, coins, buttons, corks, pegtops, sponges, chestnuts, nuts, balls used in play, reels of cotton, pieces of wood, cord, children's shoes, etc. They are usually swallowed accidentally when fetching and carrying, or when playing; but may also be swallowed unintentionally during mastication. For instance, while a dog was fetching a grape-shot ball which weighed more than  $\frac{1}{2}$  lb., he swallowed it. We once treated a dog which used to fetch coins, and swallowed a two-shilling piece on three different occasions. It is a strange fact that if needles, which are swallowed, do not stick in the oral cavity or pharynx, they rarely cause any harm. As an experiment, Zoja made cats swallow needles, some of them with the points foremost. Out of 127 swallowed needles, only two stuck in the alimentary canal; one just above the pylorus; the other in the rectum. Of the remaining 125 needles, some were passed out with the dung in from 4 to 140 hours; and others were found in the large intestine without having done any injury to the stomach or intestines of the cats which had swallowed them, and which were killed, so as to make this investigation. Similar instances have been seen in mankind. For instance, a sempstress swallowed 1,000 needles without injury.

**Symptoms.**—These are generally of a vague nature. At first, as long as the bodies lie free in the stomach, no morbid disturbance is usually noticeable. For instance, Nichoux saw a dog in whose stomach a four-shilling piece and a penny remained without any inconvenience for 12 years, at the end of which time he accidentally died, and these coins were found inside him. In other cases, acute gastric disturbance is at first set up, and, later on, if the foreign body remain for a long time in the intestinal canal, it may produce a chronic gastro-intestinal affection. If the swallowed body become impacted in the stomach or intestines in such a manner as more or less

completely to occlude the passage, the symptoms naturally become more grave. There is usually no fever at first, although the dog becomes peculiarly depressed in spirits, he lies down a good deal, eats little, and drinks often and copiously. These symptoms become complicated with vomiting, which frequently takes place with a certain regularity as to the time after the consumption of food or water. It is often performed with extraordinary violence and persistence, in which case, mucous, bilious and even faecal matters are thrown up. Well-marked jaundice is not uncommonly observed. Defecation is more or less completely suspended. Fever appears later in varying degrees. Appetite is entirely suppressed, and emaciation rapidly sets in. Symptoms similar to those of rabies are sometimes seen. The patient becomes irritable; tries to hide himself; has convulsions and epileptic spasms; runs about excitedly and aimlessly; and may manifest a strong desire to bite and a mania for destruction. Towards the end, rapidly increasing debility sets in; the expression of the face becomes plaintive; the dog remains persistently recumbent and dies comatose.

**Diagnosis.**—The history of the case and palpation of the posterior part of the body are more important for diagnosis than the symptoms we have described. Palpation should never be omitted, because it enables us in the majority of cases to determine the presence of a foreign body in the stomach or intestines. The exploration of the stomach is best effected by making the animal stand on his hind legs, and pressing on the posterior abdominal wall behind the ensiform cartilage and that of the intestines, by making the patient stand on all four legs, or by placing him on his back, and pressing on the abdominal wall.

**Duration.**—The duration of the disease is very variable, and may end at any time from a few days to several months, according to the degree of obstruction set up. We may occasionally observe a series of improvements and relapses, according to the course taken by the foreign body. Needles, in particular, show an inclination, after having perforated the wall of the intestinal canal, to wander through the body and leave it, by the formation of superficial abscesses on the ribs, flanks, tail, etc.

**Post-mortem appearances.**—If the foreign body be impacted in the pylorus, the right half of the stomach will be distended, and its muscles more or less hypertrophied, according to the duration of the disease; in fact, we have found them two-fifths of an inch thick in a medium sized dog. In such cases, the intestines will be nearly, if not quite, empty. If the foreign body has stuck fast in the small intestines, the portion of the intestine which is in front of it will be dilate and hypertrophied, and the rear portion narrowed and empty. At the site of the impaction the mucous membrane will be inflamed or will have a necrotic scab resembling tinder in appearance, which changes may sympathetically appear in other membranes, including the peritoneum. The way by which the body has travelled is marked by ulcers, scabs, extravasations of blood, pigmentation, etc. Sometimes the arrest of the body is caused, not so much by its large size, as by the irregularity of its surface (as in corks), which leads to paresis of the intestinal muscles, and the body, therefore, remains at a certain spot.

**Therapeutics.**—Treatment should commence with the administration of emetics or purgatives. As long as foreign bodies, such as coins and stones, remain in the stomach, they may often be promptly removed by an emetic (apomorphine subcutaneously, for instance); and, if in the intestine, by a cathartic, unless they are impacted, in which case, gastrotomy or enterotomy will be needed. These operations, which have been successfully performed by many veterinary surgeons, should be more frequently practised than has hitherto been done, because they entail but little danger on dogs when properly carried out. The prognosis of laparotomy is naturally unfavourable when once the foreign body has set up necrotic inflammation in the intestine.

#### GASTRO-INTESTINAL CATARRH OF PIGS.

**Etiology.**—The causes of gastro-intestinal catarrh in pigs are the same as in dogs. This disease sometimes occurs epizootically (infectious gastritis). It mostly occurs during wet seasons, and among pigs at grass; and is caused by chill, inordinate consumption of snails and worms, or by infection.

**Symptoms.**—The chief symptoms are: impaired appetite, vomiting, restlessness resembling that due to colic, constipation, diarrhoea, fever, coldness of the extremities and

especially of the ears, dry and hot condition of the snout, congestion of the visible mucous membranes, digging and hiding in the litter and drooping of the tail. The same symptoms are to be seen in "infectious gastritis," which usually runs a slow, chronic course, and often ends in death from gradually increasing prostration and emaciation. Marks described an epizoötic gastro-intestinal catarrh of pigs, in which the symptoms were loss of appetite, fever up to 105.4° F., constipation at first and watery diarrhoea later on. All the swine recovered in eight days.

**Therapeutics.**—At first the treatment is somewhat similar to that in dogs, with the exception that the doses are larger, and that drenches should be avoided, because they are difficult to give, and are liable to set up drenching pneumonia. The medicine should usually be administered in the food; or as an electuary, enema or subcutaneous injection. In all cases of impaction of the stomach, or of spoiled, bad food having been eaten, treatment should be commenced with an emetic. We generally use veratrine subcutaneously ( $\frac{1}{4}$  grain in  $1\frac{1}{2}$  dr. of spirit brings on vomiting within an hour). Hydrochlorate of apomorphine is ineffective as an emetic in pigs. Ipecacuanha and white hellebore powder are not always easy to give by the mouth; but a decoction or infusion of hellebore (15 grains in 2oz. of water) may be given as an enema. In constipation, we may give calomel (15 to 60 grains), sulphate of magnesia, Carlsbad salt, and copious enemas of soap and water. For diarrhoea we may try 15 to 30 grains of opium. Easily digestible and cooling food, such as sour milk and gruel, should be provided.

## AVIAN GASTRO-INTESTINAL CATARRH.

(Diarrhoea and Constipation.)

**Simple Gastro-intestinal Catarrh and Diarrhoea of Birds.**—This disease must be clearly distinguished from infective diarrhoea (fowl cholera), disease caused by gregarines (gregarinosis), and severe inflammation of the intestine, as in cases of poisoning. It is found most frequently in young birds of fancy imported breeds, and especially during spring, while the young birds are moulting. As in other domestic animals, its causes are errors of diet and chill. Diarrhoea occurs secondarily from entozoa, tuberculosis, etc.

The chief *symptoms* are: Diminished appetite; ruffled plumage; depression of spirits; slow evacuation of the crop; the passage, at first, of soft, white, slimy faeces, which later on become watery, and of a greenish white colour, and soil and stick together the feathers near the arcus, eroding the inner and outer surface of the cloaca; increased thirst, emaciation and weakness; and finally, if the diarrhoea does not stop, death ensues from exhaustion or haemorrhagic intestinal inflammation. On *post-mortem examination*, we find signs of desquamative intestinal catarrh, congestion, swelling, degeneration of the epithelium of the mucous membrane or inflammation of the intestine. At first the *treatment* should be diatetic. Above all things, the birds should be kept warm, and the fowl-house clean. The food should be easy of digestion, and therefore should be boiled. We may combat the diarrhoea by giving parched barley, rice water, old biscuits, hemp seed, blue poppy seed, and in the case of parrots, small pieces of chocolate, bread moistened with spirits, a teaspoonful of port wine, or bread or grain soaked in port wine. Zürn recommends oatmeal (a tablespoonful to a pint of water) or linseed tea (1 to 20 of water). As regards styptics, we generally use a watery solution of sulphate of iron (1 per cent. in drinking water). Opium is also very effective with birds, and may be given in the form of laudanum (fowls and parrots, 1-5 minims; pigeons,  $\frac{1}{2}$  minim). In very obstinate cases, Zürn gives fowls one or two teaspoonsfuls of a 1 per cent. solution of nitrate of silver daily. For simple digestive disorders, he gives minced onions or garlic, and a few pepper-corns made up with butter into pills.

*Constipation of Birds* may be caused by previous diarrhoea, entozoa, stones, feathers and other foreign bodies in the stomach and intestines; dry dung in the cloaca; digestive inactivity of the intestinal canal, etc. *Treatment* consists in giving green food, slimy and oily substances, cold water enemas; and with small singing birds, in passing into the rectum a bulb-pointed probe smeared with castor oil, and scooping out the faeces which have collected in the cloaca. The best aperients are castor oil (2 tablespoonsfuls for a strong hen), senna leaves (15 to 20 grains), rhubarb (3 to 7 grains, made into pills with honey), and calomel ( $\frac{1}{2}$  to  $1\frac{1}{2}$  grain). To small cage birds we give  $\frac{1}{2}$  per cent. watery solution of artificial Carlsbad salt as drinking water.

## COLIC IN HORSES.

**Definition.**—The term colic (*enteralgia*) is usually applied to various morbid conditions of the stomach and intestines, of which pain is the chief symptom, and which generally give rise to suppression of intestinal peristalsis. Hence, it does not signify any special disease. Colic, like broken-wind, dropsy, jaundice, etc., has a wide, collective meaning. Besides "true colic" (pain in the stomach and intestines), we have so-called "false colic," arising from painful affections of the kidneys, bladder, liver, uterus and other organs that lie in the abdominal cavity, and also certain general affections, as for instance, petechial fever and anthrax, which show secondary symptoms of colic.

The name "Colic" has kept its place in text-books of special pathology because of its long continued employment, and because horses are peculiarly liable to pathological conditions that produce pain, and of which it is often the only diagnostic manifestation.

**History.**—Colic is one of the oldest known equine diseases. Its literature can be traced back to Columella (1st century after Christ), who described three kinds of colic. Eumelus (3rd century) alluded to saltpetre as a remedy for colic. In the 4th and 5th centuries Vegetius, Apsyrtus, and Hierocles wrote about the symptoms of colic with a fair amount of accuracy. Vegetius, in particular, describes various kinds of colic, such as *dolor ventris*, *dolor coli*, *et dolor vesicae et ileus*. In the 13th century Jordanus Ruffus separated flatulent colic from colic caused by over-feeding. In the 16th century colic is fully described, especially in the writings of Vegetius Renatus and Joannes Ruellius. The works of Ehrmann, Sander and Weber appeared in the 18th century, and those of Ribbe and von Tennecker in the first half of the 19th. Among recent writers of monographs on this subject we may mention the names of von Ullrich, Legrain, Bollinger, Friedberger and Reeks.

**Statistics.**—Colic is the most important of all equine internal diseases. Out of about 70,000 cases of illness, which occur yearly among the horses of the Prussian army, about 3,000 are those of colic. Bollinger reckons that out of a total

number of 400,000 horses in Bavaria, about 40,000 are attacked yearly by colic ; and he states that the mortality is about 12 per cent. (1,625 fatal cases in 12,857). Among the 26,253 cases of colic which were reported to us by different veterinary colleges during ten years there were 2,832 fatal cases, which give a percentage of about 11. In the Prussian army the average mortality is 12 per cent. According to Bollinger, 40 per cent. of deaths occur from colic.

In the hospitals of veterinary colleges the mortality from colic has been, on an average, about 14 per cent, and in the Prussian army about 12 per cent.

**Predisposing causes.**—Horses, on account of the anatomical nature of their stomach and intestines, are more liable to colic than any of the other domestic animals. We must here bear in mind that the equine stomach is very small and, even when filled, does not reach the abdominal wall ; the oesophagus enters the stomach obliquely, and is furnished with a regular sphincter muscle ; and the stomach possesses a well-developed cul-de-sac. These peculiarities as a rule prevent vomition and the removal of solid, liquid and gaseous substances by the mouth. The great mesentery is so long, and the large intestine so voluminous and so free to move about, that it can readily become displaced or impacted with food. Also the terminal ends of the intestinal nerves, like those of the peritoneal nerves, are so sensitive to external influences, that colic may be set up without any apparent cause. And finally, aneurism of the anterior mesenteric artery, which is found in most horses, is a predisposing cause of colic, in consequence of thrombosis and embolism of the intestinal arteries.

**General causes of colic.**—1. *Chills*, which may be caused by external influences, or by consumption of frozen food or very cold water.

2. *Over-feeding*, especially when the quantity consumed is out of proportion to the amount of work, as often occurs on Sundays and holidays. The effect of over-feeding is intensified by the fact of the fodder being difficult to digest, as in the case of straw, too short-cut chaff, new corn, new hay, raw potatoes, maize, rye, beans, peas ; by sudden change of food ; by working the animal directly after feeding ; and by

## *COLIC IN HORSES.*

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the food being imperfectly masticated, owing to defective teeth.

3. *Spoiled, mouldy, fermenting and soiled food.*

4. *Formation of gas in the stomach, produced by eating young or decayed clover, especially red clover, lucerne and sainfoin, decaying grasses, rank vegetation, new grains, potato leaves, etc. Fermentation is often set up by drinking water soon after feeding. The swallowing of large quantities of air when wind-sucking may also lead to colic.*

5. *Accumulation of food in the intestines, as may occur from too long continued rest; feeding on too much innutritious food, such as straw or husks, or on too dry food; and from relaxation, dilatation or paralysis of the intestines.*

6. *The formation of calculi and concretions, and the accumulation of large quantities of sand and earth in the stomach and intestines, as often happens in campaigns and during military manoeuvres, and with horses at grass. Concretions are sometimes formed in the intestines of the horses of millers and bakers by long continued feeding on bran and sharps, both of which contain a large amount of phosphate of magnesia.*

7. *Intestinal worms, especially collections of *ascaris lumbricoides*.*

8. *Intestinal stenosis from compression, displacement, volvulus, invagination and incarceration of the intestine.*

9. *Long fasting (hunger colic), which occurs chiefly among military horses in war time.*

10. *Rolling produces colic in rare instances by displacement of the intestines. Thus, Köhne saw a case of volvulus caused by a horse being rolled on his back when cast for an operation. It is a well-known fact that recent twists of the guts are found at slaughter houses in pigs and calves which have been rolled about a good deal during unloading. On account of these exceptional cases, it would be unreasonable to prevent the rolling of a horse which was suffering from colic. Experience shows that horses at liberty roll with impunity, and that recent volvulus has often been cured by rolling.*

11. *Colic is met with in certain diseases of the intestinal tissues, such as catarrh, inflammation, croup, ulcers and poisoning.*

**Symptoms.**—The most prominent symptom is pain, which may come on suddenly or gradually. If in harness or saddle, the horse goes "short," gives way in his hind-quarters, stops,

becomes restless, paws the ground and stamps, shakes himself, and can be prevented only with difficulty from lying down. In the stable the true pains are sometimes preceded by depression of spirits, loss of appetite and hanging back from the manger. Later on, he often totters, groans, kicks with the hind legs at the abdomen, looks round at his hind-quarters, whisks his tail, stands over at the knees, arches his back, stretches out the head and neck, and places the hind feet under the body. As a rule, the animal lies down very carefully and slowly, or makes attempts to lie down, but sometimes may throw himself recklessly on the ground. When he has got down, he may lie quietly, or may throw himself about, roll and kick ; or he may remain quietly on his back, with the legs drawn towards the body, for a long time. The pains may temporarily disappear, and the horse get up and begin to feed again, but the state of uneasiness will soon return.

The bodily temperature is unequally distributed, and the ears and extremities are usually cold. Sweat often breaks out, especially at the base of the ears, on the sides of the neck and ribs, belly and flanks, which places are sometimes hot, at other times cold, and occasionally the sweat drops on the ground. The visible mucous membranes are usually red, or may be discoloured in various ways. The oral cavity is very dry and hot, and its mucous membrane sometimes affected with catarrh ; or it may be very cold and pale, with a washed-out appearance. The abdomen is tense, and may be more or less distended with gas, but it is tucked up only on rare occasions. Usually peristalsis is greatly diminished or entirely suppressed, and very faint, if any, intestinal murmurs are heard on auscultation ; but in exceptional cases loud metallic sounds are heard. Defecation is delayed and the faeces, as a rule, are dry, hard, formed into small balls, covered with mucus and occasionally bloody. In many cases, only a few balls of dung are pressed out by great straining. Sometimes the faeces are very soft and even watery, are of an acid smell, contain undigested particles of food, and flatus frequently escapes. Occasionally necrotic and desquamated pieces of the intestine are found among the faeces.

On exploring the rectum, we may find it completely filled with dung, quite empty, or containing only a few balls of faeces. The mucous membrane is hot and swollen ; the neighbouring portions of the intestine are often firm and tense to the touch, in consequence of their being filled with excrement or gases, and the bladder may be excessively filled. The lumen of the

rectum may be greatly dilated, or so much narrowed, that we may be unable to pass even one finger through the constricted part. Examination of the inguinal rings in stallions may reveal a strangulated hernia. When the colon is abnormally distended, we may feel one of its longitudinal bands in the form of a tensely stretched cord in the abdominal cavity. In rare cases we may find a calculus in the rectum. Micturition is usually delayed, although the animal often stretches himself out, draws his penis and tries to stale, but as a rule is able to pass only a few drops of urine, which may be clear in colour, or of such a rich tint that it may appear to contain haemoglobin. It may be of an alkaline, neutral or acid reaction and now and then contains albumen.

The breathing is generally laboured and often very difficult. The pulse at first suffers no great change, but later on its rate increases to 50 or 60 beats per minute and, in extremely grave cases, to 100 or more. The heart-beat becomes stronger, and the cardiac sounds are often very loud. The temperature is normal at the beginning of the attack, but may rise to a high degree during the further progress of the disease, although the fever may abate to a great extent shortly before death. The animal's mental faculties are more or less affected, and any bad habits which he may have, like crib-biting, are discontinued.

In graver forms of colic more severe symptoms are seen. The animal assumes peculiar attitudes; for instance, he kneels down, while keeping his hind-quarters raised; lies on his sternum with his fore-legs stretched out (in a position similar to that of the Sphinx); sits like a dog, namely, on his hind-quarters with the fore-quarters raised. The animal eructates, stretches himself out, retches and may vomit, in which case the vomited matter is often discharged through the nostrils. The pain may increase to such a degree that the animal grows mad, rears up, snorts, groans, screams, rages, may even attack men, bite the manger or parts of his own body, grind the teeth, or may press forward as if stunned; circle round and round always the same direction; make nodding, jerking and pendulous movements of the head; lean senseless and stupefied against the wall; tremble, become convulsed and covered with a cold and profuse sweat. The pulse becomes thready, intermittent or imperceptible; the heart-beat tumultuous; the expression haggard; the pupils dilated; the lower lip pendant; the rectum gaping and sometimes prolapsed, and a brownish fluid flows

from the anus. Finally, the animal staggers, falls on the ground, becomes convulsed and dies. Some he neighs loudly before dying and may occasionally drink a large quantity of water. At other times the pain suddenly ceases, and coma and death speedily follow.

**Course and Duration.**—In the large majority of cases the course is short and acute. Occasionally the symptoms of colic disappear in a few minutes, but usually they continue for a few hours; their longer persistence than thirty-six hours is a very unfavourable sign. Colic may, however, take a subacute or chronic course, in which case it may last for a fortnight, and with intermissions for several weeks, especially when the malady is due to constipation.

The chief resulting complications of colic are suppurating superficial sores caused by pressure on bony protuberances which are thinly covered with skin, such as the orbital arch, zygomatic arch, edge of the lower jaw and anterior iliac spine; and injuries caused by the animal throwing himself down and rolling about.

**Prognosis.**—The prognosis of colic should always be made with reserve.

*Signs of improvement are:*—Abatement of pain; a more uniform distribution of bodily temperature; cessation of sweating; improvement in the condition of the pulse; evacuation of faeces and flatus; return of appetite, desire for water, etc.

The following are *unfavourable signs*:—Symptoms suggestive of gastro-intestinal inflammation, peritonitis and rupture of the stomach, intestine or diaphragm, which symptoms are generally as follows: hard, small and very frequent pulse; very high or sub-normal temperature; extreme pain; sudden abatement of great pain; cold sweats; great coldness of the extremities; persistent constipation; complete suppression of peristalsis; great distention of the abdomen; dyspnoea; vomiting; staring and glassy expression of the eyes, etc.

**General Differential Diagnosis.**—True colics, which are caused by pathological conditions of the stomach and intestines, may be confounded with a large number of painful affections in other organs, such as:—

I. *Rheumatic Hæmoglobinuria*, the symptoms of which are often very similar to true colic; as, for instance, its sudden

appearance after a chill, manifestations of intense pain, cessation of peristalsis, suppression of defecation and micturition, violent outbreaks of sweat, dyspnoea and derangement of circulation. It is probable that, on this account, rheumatic haemoglobinuria has been frequently mistaken for colic. Close examination will, however, give us ample data for their differentiation. In true colic there is no paralysis of the hind limbs, no pain nor swelling of their muscles, which are characteristic symptoms of the other disease. The urine also contains neither haemoglobin nor dissolved blood. In true colic the animal often lies down and gets up again; but in rheumatic haemoglobinæmia the patient is generally unable to rise.

2. *Labour pains.*

3. *Retention of urine* from paralysis or obstruction, which is often mistaken for ordinary colic by laymen, because the pressure exerted upon the bladder by the distended intestine excites the animal to make efforts to urinate, even when the bladder is empty. Examination of the bladder by the rectum, or the introduction of a catheter, will give the necessary information. Retention of urine, which is rare in horses, does not interfere with the activity of the digestive organs.

4. *Inflammation of the Bladder.*—Here we obtain diagnostic data from the condition of the urine, which contains vesicular (transitional) epithelial cells, pus corpuscles and crystals of phosphates; sensitiveness of the bladder to pressure from the hand passed into the rectum; and frequent passing of urine in drops, in addition to the fact that the gastro-intestinal canal is unaffected.

5. *Metritis* may be distinguished from colic by the fact of a preceding birth, the usual presence of a discharge from the vagina, abnormal sensitiveness of the uterus and longer duration of the disease.

6. *Displacement of the Uterus* (torsion, prolapse, inversion and invagination). With internal and external examination only incomplete prolapse will present any difficulty in diagnosis.

7. *Strangulated hernia* always gives rise to symptoms resembling those of extremely severe colic, but can be recognised by a careful examination of the inguinal rings and scrotum in entire stallions and geldings.

8. *Peritonitis.*—It is always difficult, and sometimes impossible, to distinguish between peritonitis and colic, especially when the latter has a rheumatic origin. In fact, peritonitis can be diagnosed in horses only in certain cases. It can be recognised

with comparative ease when it is secondary, as after castration, laparotomy and herniotomy; or when it follows inflammation of the kidneys, bladder or uterus. The peritonitis which may occur during colic can often be recognised by the height of the accompanying fever, the smallness and frequency of the pulse, and by rapidly increasing prostration.

9. *Nephritis*, which is diagnosed chiefly by the presence in the urine of red and white blood-corpuscles, epithelial cells of the kidneys, urinary casts and albumen. Moreover, in nephritis the colic symptoms are much slighter, and consist usually in a greater sensitiveness in the region of the kidneys and loins and in stiffness of gait.

10. *Inflammation of the Liver*.—Acute hepatitis, which is exceedingly rare in horses, manifests itself in slight restlessness, which is probably traceable to inflammatory irritation of the peritoneal covering of the liver. If no distinct icterus be present, it might be difficult to make a correct diagnosis.

11. *Intestinal Catarrh and Inflammation, Ulcers*.—Among the various pathological conditions of stomach and bowels those most productive of colic pains are catarrh and inflammation of the mucous membrane, and also ulcers. Thus we speak of colic caused by inflammation and also by indigestion. As, however, both catarrh and inflammation or ulceration of the gastro-intestinal mucous membrane will be discussed as independent diseases, the attacks of colic occurring during their course must be distinguished from true colics, although by no means always easy to do. The following points may perhaps be worth mention:—In intestinal catarrh the colic symptoms are rather of a secondary character and tend to disappear quickly, but are usually accompanied by diarrhoea; the disease itself is slighter and more lasting. In case of inflammation of the intestine, attention must above all be paid to the history of the case, whether perhaps irritant substances or poisons (so-called "poison-colic") may not have affected the intestine. Thus excessive and sometimes bloody diarrhoea, while the posterior part of the abdomen is empty, with high fever, and following on a previous persistent constipation, indicate rather an inflamed condition of the intestinal mucous membrane than another form of colic. Ulcers in the stomach or intestines, it is true, are difficult to recognise with certainty; they may, however, be distinguished by their chronic course and by occasional haemorrhage, from colic which takes on the whole an acute course.

12. Finally, various diseased conditions of the body may be

mistaken for colic, if accompanied by acute pain; but careful examination will always prevent such errors. Such conditions are: inflammation of the brain, pleuritis, pneumonia, petechial fever, influenza, painful surgical affections, especially inflammation of the hoofs or joints, and, in certain cases, also thrombosis of the iliac and femoral arteries.

**General Anatomical Notes.**—From *post-mortem* examination we find that the phenomena of colic vary very much according to its cause. Volvulus of the colon and small intestine, as well as laceration of the stomach are most frequent, and occur pretty uniformly in the ratio of one in five of all fatal cases.

Of 1,882 *post-mortem* examinations made in the Prussian Army during five years, 347 cases, or 18 per cent. of the whole, were found to be cases of stomach-laceration; 336, or 17 per cent., were volvulus of the colon; 331, or 17 per cent., were volvulus of the small intestine. Thus the above-mentioned three pathological changes were found in about half the entire number of cases. Next in frequency came gastro-intestinal inflammation and laceration of the colon (each 8 per cent.), also laceration of the diaphragm with prolapse of parts of the intestine (5 per cent.). Less frequent were laceration of the omentum or mesentery, with imprisonment of portions of intestine in Winslow's foramen, obstruction of the colon and volvulus of the blind-gut (3 per cent.). Bowel-stones were rare, as also was compression of the small intestine by lipomata, laceration of the same or of the rectum, invagination of the small intestine into itself or into the blind-gut or cœcum, diverticula of the intestines and obstruction of the small intestine (each from 0.5 to 1.5 per cent.). Volvulus of the rectum, invagination of the rectum into itself, worms, etc., occurred only in isolated cases as the cause of death.

With regard to these changes we would remark as follows: In displacement (volvulus) of the intestines the twisted or knotted part of the bowel is generally very pale or anaemic, while the walls of the intestine, both in front of and behind the affected spot, are swollen and infiltrated with blood, the mucous membrane is dark red and tender, the contents of the gut are bloody and the mesentery is gelatinous and charged with blood. Collections of foodstuff, stones and concretions all lead to catarrhal, haemorrhagic and diphtheritic or necrotic inflammation of the mucous membrane. They chiefly occur at the

junction of the ileum and cæcum, and at that of the colon and rectum, and especially in the blind-gut or cæcum. Twining of the small intestine around the colon, or some other portion of itself, occurs sometimes. We also find the intestines twisted through lacerations in the omentum, strangulated by pedunculated lipomata and other new growths on the mesentery, and compressed by overloading of neighbouring intestines. Constriction of intestinal loops occurs in the inguinal canal, in some old-standing rent of the diaphragm, in the foramen of Winslow, in crevices of the omentum, the mesentery or of the broad uterine ligament, and between ribbon-like, old peritonitic adhesions. The intussusception most frequently met with is that of the small intestine into the cæcum. Rupture of the stomach generally occurs along the great curvature, at the spot where the serous covering is continued into the layers of the omentum, and may extend to three-fourths of its length. The edges of the rupture are inflamed, swollen and charged with blood, serrated and irregular. The rent in the serous covering is usually longer than that in the mucous membrane. Food is found lying between the several layers of the wall. The contents of the stomach are partly bloodstained, and penetrate into the abdominal cavity. Laceration of the intestine usually takes place in parts where faecal stasis has occurred. Sometimes only the mucous membrane is torn, and the intestinal contents penetrate beneath it, so that it is raised from its support to a considerable extent; or the contents burst through the muscular wall on the side of the mesentery and penetrate between the mesenteric layers. Ruptures of the diaphragm show very ragged edges, between which a part of the stomach or bowels projects into the thoracic cavity.

The intestinal mucous membrane is sometimes hyperæmic, at other times inflamed, the inflammation being either of a catarrhal, croupy, diphtheritic or haemorrhagic nature. Occasionally it is necrotic and ulcerated. The sub-serous and sub-mucous connective tissue is infiltrated with blood, the contents are also bloody, the intestinal walls swollen, tender and friable. The peritoneum exhibits the utmost variety of inflammation from the sero-fibrinous to the malignant and ichorous. In the mesentery are numerous haemorrhages. The mesenteric glands are swollen and charged with blood. Very frequently the anterior mesenteric artery is aneurismal, and contains the larvae of *Strongylus armatus*, while in the intestinal arteries are numerous thrombi and emboli. The

general changes consist in hyperæmia of the abdominal organs and also of the lungs (the latter from overcharging), œdema of the lungs, hypostatic inflammation of the lungs, haemorrhages of the pleurae and meninges, parenchymatous degeneration of the heart, kidneys, liver and spleen, and, lastly, in the presence of dark, almost black, tar-like blood.

**General Remarks on Causes of Death.**—If, on dissection of a fatal colic case, a gastric or intestinal rupture, an extensive enteritis or peritonitis be found, there can then be no doubt as to the cause of death. But very often a case of colic may end fatally within twelve to twenty-four hours, without any such changes having in so short a time been produced in the abdominal cavity as would explain so speedy a fatal issue. The following causes of death are all possible in such a case.

1. *Absorption of toxic material into the blood.*—This may either be of a chemical or septic nature, or a ptomaine or leucomaine poison. It has already been proved by experiment that in the fluid contents of an imprisoned hernia, micro-organisms can be detected long before necrosis of the intestinal wall sets in. They arise from the interior of the bowel, for both bacilli and coccidi penetrate the wall of the intestine and produce fatal peritoneal sepsis, and generally without any accompanying suppurative peritonitis. The liver seems in such cases to be the most important transitional depot for the septic matter. This organ is found to be in an extreme state of septic corruption, sometimes even immediately after death, and before natural putrefaction could have commenced.

2. *Compression of the lungs, followed by œdema, with suffocation.*—Caused by pressure of the distended intestines upon the diaphragm. The proof of this lies in the frequent detection of œdema of the lungs and other symptoms of suffocation at post mortem examinations.

3. *Blood poisoning by carbonic acid in tympanites.*—This occurs when the pressure (or tension) of the carbonic acid in the blood falls below that of the intestinal gases (which consist chiefly of the same acid); for then, in accordance with strictly physical laws, the blood absorbs carbonic acid.

4. *Increase of arterial blood pressure, with consecutive brain and lung apoplexy,* consequent upon the pressure of the distended bowels upon the abdominal aorta and larger veins.

In addition to these causes, which may all certainly prove fatal, other explanations were formerly sought for. Thus heart-shock caused by

extreme pain was held to cause death, which has little probability to command it. Phenol poisoning of the body, through absorption of large quantities of phenol developed during abnormal bowel fermentation, was also thought to be a cause of death. But Tereg has proved that during colic the absorption of phenol is rather diminished than otherwise. The commonest causes of a fatal ending may be sought, therefore, in sepsis, oedema of the lungs and blood-poisoning by carbonic acid.

**General Treatment.**—The first provision requisite for a horse suffering from colic is a wide and spacious enclosure in which the animal can fling itself about without injury, and a plentiful supply of dry straw. Then the body of the animal should be rubbed all over with straw and woollen rags, in order to stimulate blood circulation and intestinal activity. This may be rendered more effective by sprinkling the skin with spirits of camphor, to which turpentine may be added. In slighter cases, when the colic symptoms are not too severe, and weather permits, one may try to stimulate the peristaltic action of the intestines by a little gentle exercise; this latter is specially advisable in cases of constipation-colic, the course of which is chronic, but all severe exertion, such as trotting or galloping, is to be avoided. In severe attacks of colic no attempts at movement should be made, as they do more harm than good.

With every colic patient an examination of the rectum is imperative, partly for removal of any accumulated dung, and partly to assist diagnosis. Further, one must endeavour to soften the faeces and stimulate peristaltic action by means of enemas. Glycerine enemas have (like soap-clysters) only a local effect, and produce no stimulus of peristaltic action in the deeper intestines.

The practice among veterinary surgeons of introducing large quantities of water into the rectum is very old, but its effects have been much exaggerated. It has been asserted that ten gallons and more may be injected, and that the water will penetrate as far as the blind gut. But the fact is that any rinsing of the intestinal canal beyond the rectum itself can seldom be effected, and that more than two gallons can rarely be retained. As for the temperature of the water thus used, it should in acute and feverish colic be *cold*, but in chronic cases *warm*. Hand in hand with this internal application of water, cold or warm fomentations of the abdomen should also be used.

Moderate rolling about can certainly not harm a horse suffering from colic, and in cases of twisted intestines may even do good. But, on the other hand, violent rolling, impetuous flinging of itself upon the ground, and, indeed, all excessive

restlessness must be controlled as much as possible, as they may cause rupture of the tightly-packed stomach or intestines. The best sedative, and therefore also a specific remedy against spasmodic colic, is an injection of morphia (5 to 8 grains). Larger doses, as for instance 15 grains of morphia, may produce undue excitement, with a wild desire to run about, and even maniacal symptoms.

If the abdomen be greatly distended with gas, do not delay to puncture the intestine, an operation which probably never causes any injury, if performed with due antiseptic precautions. Blood-letting is also sometimes necessary if apoplexy of the lungs or brain be threatened. Internal administration of so-called gas-absorbing remedies (spirits of ammonia, lime-water, sulphuretted potash and hyperchloride of sodium) is but of doubtful effect.

For diminished peristaltic action and delayed evacuation, many remedies are in use. In general they are the same as have already been cited in connection with constipation which accompanies intestinal catarrh. Sulphate of eserine ( $\frac{1}{2}$  to  $1\frac{1}{2}$  grains) given hypodermically in watery solution, as recommended by Dieckerhoff, is deservedly highly spoken of. The use of eserine must not, however, be indiscriminate, for it is contra-indicated in cases of spasmodic colic, because of its tetanic and spasm-producing effect; also in cases of acute colic produced by over-feeding, in wind colic and in graver cases of colic arising from constipation, on account of the danger of stomach or bowel rupture. On the other hand, it is stated that since the introduction of eserine, the cases of twist and volvulus of the small intestine have increased in number. It is therefore wiser to adhere to the smaller doses ( $\frac{1}{2}$  to 1 grain) rather than to the larger (1 to  $1\frac{1}{2}$  grains). As moreover a few instances of poisoning, as well as of abortion in pregnant mares have occurred (due probably to the inconstant nature of the drug), it is wise to use eserine with caution. Especially with horses suffering from lung disease is this advisable, owing to the risk of oedema. A combination of eserine ( $1\frac{1}{2}$  grains) with pilocarpine (3 grains) is suggested by Ellenberger. The object of the latter is to stimulate bowel secretions, and to liquefy the contents of the intestines. As a substitute for eserine, which is easily decomposed and very inconstant, and for pilocarpine, which is costly, Fröhner recommends hydrobromide of arecoline in subcutaneous doses of  $\frac{1}{2}$  to  $1\frac{1}{2}$  grains. Dieckerhoff has lately recommended barium chloride in doses of  $7\frac{1}{2}$  to 20

grains to be injected into the veins, or in doses of 1 to  $2\frac{1}{2}$  drams, if per orem.

Glauber's salt and tartar emetic have long been used, the former in 8 to 16 oz. doses, and the latter in 1 to 2 drams. They are often combined. The following are also in common use :— Aloes, 4 to 8 drams; calomel, 1 to  $2\frac{1}{2}$  drams; castor oil,  $\frac{1}{2}$  to 1 pint; linseed oil, 1 to  $1\frac{1}{2}$  pints; and in obstinate cases croton oil, 15 to 20 drops is used occasionally.

The above-mentioned drugs are not required in every case of colic. Indeed, it is a well-known fact that a moderately large percentage of colic patients recover without any medical treatment. It would, however, be a mistake to infer that in colic a waiting policy is always best.

We cannot endorse the uniform treatment of all colic cases with one and the same remedy, such as Lemke and Eckl recommend (viz., morphia), Adam (cold water douches), Luelfing (a mixture of aloes, Glauber's salt and fenugreek). Every case requires individual treatment. Next to subcutaneous injection the best method of administering the medicines named is by means of electuaries and balls. Drenches can do no harm if properly administered, that is, by the surgeon himself. But when used by the owner there is risk of pneumonia arising from the passage of the liquid into the trachea.

#### I. SPASMODIC COLIC.

*Simple, Inflammatory, Nervous, Rheumatic and arising from Colds.*

**Definition.**—Spasmodic or cramp colics are those caused by internal or external chill, and which cannot be traced to any change of the tissue, or other material cause beyond a painful contraction of the bowel. Seeing that horses are much exposed to cold, colic is naturally not rare amongst them, especially in spring and autumn. In breeding establishments, among the nobler and finer-skinned horses, this is by far the commonest form of colic, as also among cavalry horses, with which—except in times of war or manoeuvres, irregular feeding does not often occur.

**Symptoms.**—The pain arising in colic independently of all intestinal changes is probably traceable to compression of the sensitive nerves of the bowels by contraction of their muscular walls. Some believe it to be the same as rheumatic muscular pain. In spasmodic colic this pain is usually rather violent, if not of very long duration. It appears

quite suddenly and in spasms, soon to abate and then reappear again with increased intensity. In addition to this, in contrast to all other forms of colic, stoppage and the cessation of peristaltic action may be absent in cases of spasmodic colic, and the bowels may in fact be unusually active and noisy, ending even in diarrhoea, owing to the spasmodic contraction of the bowels. The so-called 'spasmodic rumbling' is very loud as compared with other kinds of colic, it sounds as though water drops were falling from a certain height upon a thin, ringing metal plate, and can be explained by the difference of tension of the gases in the several bowel sections, causing the gas to gush with small explosions from the highly charged loops into those less densely filled. Spasmodic colic generally passes off after only a few hours, sometimes even in  $\frac{1}{2}$  to  $\frac{1}{4}$  an hour. But when it lasts more than 12 hours, it may be followed by serious complications, and especially volvulus.

**Differential Diagnosis.**—Here we have especially to guard against confounding spasmodic colic with the embolic form. Among the large number of so-called nervous or rheumatic colic cases which recover, some are certainly of embolic nature, as, owing to their similarly sudden and intermittent appearance and the impossibility of positive diagnosis by operation, an exact differentiation is impossible. But this confusion cannot arise to the extent asserted by Bollinger, seeing that spasmodic colic is usually only supposed to exist when previous chill can be proved, while in embolic or thrombotic colic the absence of all external cause is a point of first importance in forming an opinion.

**Therapeutics.**—In the treatment of spasmodic colic the so-called "anti-spasmodic" remedies have always played a chief part. Of these morphia stands first. Whilst the indications for its use are in all other kinds of colic principally symptomatic, they are here especially causal, not to say essential. In many cases, therefore, morphia alone suffices. It not only removes the spasmodic and painful contraction of the bowels, but also the serious danger of twisting of the intestines. The subcutaneous dose of hydrochlorate of morphia is 5 to 7 grains in a 4 per cent. watery solution. The colic pains and bowel cramp usually subside quickly, so that a second injection is rarely necessary. The other nervine

sedatives, such as belladonna, hyoscyamus, aconite, ether, chloroform, etc., have become superfluous since the introduction of morphin.

The usefulness of warm infusions made from camomile, peppermint, valerian and elder-flowers cannot be disputed, as, owing to the ethereal oil they contain, they have a certain undoubted sedative power. Most ethereal oils, owing to their evaporation in the intestinal canal, produce an anti-spasmodic effect on the surrounding organs, reducing their reflex excitability. Nevertheless the administration of ethereal oils in the form of infusions should be only rarely adopted, owing to the dangers which accompany drenching, and because of their much slighter effect as compared with morphia. The use of physostigmine is contra-indicated.

As regards external treatment, we recommend vigorous rubbing down with turpentine and spirits of camphor (1 to 10-20), and warm coverings. Cold wrappings and enemas are not advisable, as they only increase the contraction of the bowel muscles. On the other hand, the application of hot water cloths to the abdomen and warm injections have a distinctly soothing effect. Finally, complete rest is much to be preferred to any movement or exercise.

## 2. COLIC DUE TO OVER-FEEDING.

**Symptoms.**—This form of colic is distinguished by violent and long continued paroxysms, and often by a very quickly fatal ending. The breathing is generally very heavy, the abdomen greatly distended, abnormal positions are common, especially that assumed by dogs when sitting, by which the animals undoubtedly seek to relieve both diaphragm and lungs from the pressure of an over-loaded stomach. Characteristic symptoms, though not very frequently noted, are—belching, retching, straining and vomiting. Not unfrequently these end in rupture of stomach or diaphragm, which is recognised at once by the sudden cessation of the colic symptoms, and general signs of collapse.

**Diagnosis.**—Here, strict regard must first be had to the history of the case, and to verification of the fact that the colic arises from too liberal feeding. Without this knowledge it might be difficult, or even impossible, to distinguish it from any other form of colic. For even the characteristic sign of

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overfeeding, viz., vomiting, may arise from other pathological conditions of the stomach or intestines.

**Prognosis.**—This must generally be made with caution, the more so, as the horse is unable, or only rarely able to relieve its stomach, as other animals do, by vomiting, and is liable to rupture either stomach or diaphragm if it throw itself down with violence.

**Therapeutics.**—The treatment of colic, arising from over-feeding, consists first of all in emptying the rectum by manual exploration, and in frequent administration of clysters, so as indirectly to expedite the advance of the contents of the stomach. In slight cases, eserine and arecoline may be given in small doses ( $\frac{1}{4}$  gr.), as well as tartar emetic ( $\frac{1}{4}$ -4 drs.) to stimulate peristaltic action. In all severe cases, eserine and arecoline must be avoided, owing to the great danger of stomachic rupture. In such instances, all one can do is to prevent the animal from plunging or flinging itself on the floor, by morphia injections (7 grs.). Dieckerhoff recommends barium chloride (7-20 grs. if injected into the veins;  $1\frac{1}{2}$ -2 drs. if in draught).

**Vomiting.**—Special interest pertains to the symptom of vomiting, which occasionally occurs in cases of colic caused by over-feeding. The act of vomiting, which, as is elsewhere explained, is peculiarly difficult for the horse, happens sometimes in these cases, owing to the abnormally distended condition of the stomach and the relaxation of the otherwise firmly closed cardia. The phenomena which have thereby been noticed are as follows: After spasmotic contraction of the belly and neck muscles, accompanied by an in-drawing of the head to the breast and lowering of the neck, a fluid is discharged from the mouth and nose, of varied consistency, according to the food consumed, and generally greenish-yellow, frothy and sour in smell. The quantity is very irregular. Sometimes a whole stable-bucketful is discharged at once, and at other times, after hours of vomiting, very little fluid is emitted. During this vomiting the animal sweats, puts its feet under the body and shows widely-opened, staring eyes. When the fit is over, it seems much exhausted, sways about and trembles and coughs for a long time. Sometimes it does not reach the point of vomiting but only slavers, at the same time straining, retching and belching. With regard to the prognostic signification of vomiting, it is a mistake to regard it as an unfavourable, or indeed ominous symptom. In many cases it undoubtedly relieves the stomach, and in very many others no injurious after-effects have been noticed. Nevertheless, it must in general be considered as a sign calling for great caution, as, with an abnormally distended stomach, rupture of that organ may easily take place; and also because other serious affections of the gastro-intestinal

canal may be the provoking cause of vomiting, such, for instance, as an ulcerated stomach, stenosis of the intestine or peritonitis. Vomiting also occurs in certain diseases of the oesophagus, especially distension as well as in inflammation of the diaphragm. Finally, vomiting also follows sometimes on the administration of ecerine, arecoline and pilocarpine.

**Rupture of the Stomach.**—This happens during the course of colic produced by overfeeding, owing to the excessive distension of the stomach and final rending of its walls; or to violent shock caused by the animal flinging itself down; or, finally, to spasmotic contraction of the abdominal walls during vomiting. It happens also when vomiting is prevented by the firmly closed cardia, whereby "auto-rupture" of the stomach is produced by its own excessive muscular contractions. On the mutual relation of vomiting and rupture of the stomach erroneous opinion were long current, for people believed that the former was the result and not the cause of such ruptures. But it is quite impossible that after the stomach has been ruptured, and its contents emptied into the abdominal cavity, any portion of those contents should find their way out through the mouth. When, therefore, vomiting and stomach rupture are found together, the latter can only be regarded as a consequence of the former. On the contrary, though, where there is only a partial rupture of the walls of the stomach, as, for instance, when only the muscular coat, or a portion of the same, is rent, this rupture may itself be a cause of vomiting, if it coincide with a paralysis of the cardia. The phenomena of bowel and stomach rupture are difficult to differentiate. Sometimes they are preceded by vomiting, retching and eructation. Generally, however, rupture shows itself suddenly in a complete collapse at the height of a violent colic attack. The signs of colic disappear, the abdominal sounds become inaudible, the pulse is feeble and extremely rapid, the extremities grow icy cold, the body is bathed in cold sweat, and the internal temperature sinks below normal or rises rapidly. The animal sways and trembles, sometimes neighs, assumes extraordinary positions, appears as though stupefied, draws its feet under the body, experiences great difficulty of breathing and shows an anxious and staring expression and a stiff gait. Sometimes a viscid, greenish-yellow mucus exudes from the mouth. Death generally occurs in a few hours, but sometimes not for several days, if the rupture be only partial, or if some organ, such as the omentum, lie across the rent. Such cases, however, are uncommon.

**Rupture of the Diaphragm.**—This arises from the pressure of an over-filled stomach, or from the animal suddenly flinging himself on the ground, and is facilitated by an antecedent hernia or inflammation of the diaphragm. On dissection one finds that either the stomach, small intestine, colon or omentum, is protruded through the opening into the thoracic cavity, and in chronic cases is sometimes found grown fast to the lungs, ribs or pericardium. In such cases the inserted portion of the bowels often becomes constricted. The symptoms of rupture of the diaphragm are, first of all, excessive difficulty of breathing, which may amount to suffocation, and in which the abdominal muscles are spasmodically contracted. The animal then sometimes has

violent fits of colic, strikes extraordinary attitudes, especially that of sitting like a dog, sways about, sweats profusely, coughs, breathes with a snore, is unable to neigh, but can only emit feeble noises. If a bowel has slipped forward into the thoracic cavity, one can hear on percussion of the thorax, frequently as far as the shoulder, a clear, drum-like sound, and on applying the stethoscope, a series of gurgling, rumbling and splashing bowel-sounds; the usual vesicular breathing sounds meanwhile being obscured. These latter symptoms render the diagnosis certain under all circumstances. Nevertheless, in rupture of the diaphragm they may be absent, as no portion of the bowels, or only very little, may have fallen forwards, occupying only the lower part of the cavity. Moreover, one must be careful not to confound any unusually loud peristaltic murmur of normally situated intestines with the above noises. The progress of a rupture of the diaphragm is generally very acute, and death from suffocation often happens in a few minutes, or within half an hour. In rarer cases it is chronic; the animal apparently recovers, sometimes quickly, sometimes after a colic lasting for several weeks. But it relapses later, often at quite regular intervals, say, for example, from four to fourteen days, or after the slightest exertion. Others are greatly distressed, particularly in going downhill, and others never lie down again. Finally, permanent difficulty of breathing and gradual emaciation may result.

### 3. COLIC FROM EMBOLISM AND THROMBOSIS IN HORSES.

**Historical Notes.**—Aneurism of the anterior mesenteric artery, caused by worms (worm-aneurism), was very early associated with colic by medical men. Thus, Rigot (1829), and Schutt (1835) discovered aneurism after previous colic. Brückmüller speaks of cases "in which aneurism of the mesenteric arteries is said to have produced the colic so common in horses," and is of opinion that a causal relationship between them cannot be denied. Prehr seeks in one case the origin of the colic in the actual obliteration of the anterior mesenteric artery. Bouley says that bloody excrements and intermittent colic are special elements in the diagnosis of aneurism of the mesenteric arteries. Köhne believes that in a colic case watched by him, and caused by stoppage of the anterior mesenteric artery, the large intestines, and especially the blind-gut, were reduced to a state of paralysis. Hahn assumes as the cause of a periodically returning colic, the aneurism of the anterior mesenteric artery revealed by dissection. Finally, Cornevin described a very fine case of thrombotic colic, with occlusion of the arteries of the ileum and cæcum.

From these particulars it is plain that the relations of cause and effect between aneurism and colic had been sought, and

at least partly found, prior to the publication of Bollinger's work, "Colic in Horses, and Worm-Aneurism of the Intestinal Arteries." Notwithstanding, great merit is due to Bollinger for having clearly established the relationship by his extensive researches.

**Anatomical Notes on Worm-Aneurism of the Anterior Mesenteric Artery.**—Hering showed in 1830 that this aneurism frequently occurred in the horse. Indeed, he maintained that a horse is more likely to have an aneurism than not. According to the calculations of Bruckmüller, Roell, Bollinger and others, 90-94 per cent. of all horses suffer from the aneurism in question. It seems to be most common in aged animals. Thus, Ellenberger only found a single horse free from aneurism among 85 used for dissection. It is met with in quite young foals of three months, though it appears not to be present in the newly born.

Aneurism of the anterior mesenteric artery, which supplies with blood all the small and large intestines, except the middle and hinder regions of the rectum, is by far the most frequent. It is rarer to find the posterior mesenteric, or the aorta affected. The walls of the aneurisms are mostly very much thickened, especially the middle coat and the peritoneal covering. In form they are usually evenly expanded to a long oval, and bottle or spindle shaped, more rarely bag-shaped, as large as a man's head, and even as much as a foot in length. The inner coat displays the most varied inflammatory and degenerative changes, such as swelling, fatty degeneration, calcification, ossification, ulceration, and sclerosis of the connective tissue. The contents consist mainly of a parietal, stratified, and often vascular thrombus, which may entirely obstruct the lumen of the aneurism, and even project itself into the various branches of the several arteries, and sometimes even into the lumen of the aorta. Not unfrequently it is very loose and friable, and at times even softened and degenerated. In addition to the thrombus a few larvae of the *Strongylus armatus* Rud. are found; on an average nine in each aneurism. The presence of these worms is the proximate cause of the chronic changes in the inner coat, and consequently of the thrombus and aneurism also, as they maintain a chronic traumatic endoarteritis by constant irritation of the intima. Their number may be very considerable (100-200 and more). As to the manner and period of their immigration nothing certain is known.

**Worm-Aneurism in its Relation to Colic.**—Apart from the fact that aneurism of the anterior mesenteric artery hinders the due nourishment of the bowels by checking the supply of blood, and thereby pre-disposes to colic, the bowel is further permanently exposed to a three-fold danger. First of all, the thrombus may entirely choke up the lumen of the anterior mesenteric artery (this is rare); or it may prolong itself into any branch of a bowel-artery; or, finally, a severed particle of the thrombus may be carried along by the flow of blood, as an embolus, into one of the intestinal arteries. In all three cases, unless sufficient collateral circulation be developed, changes are set up, as has been abundantly demonstrated by experiment. First, then, an arterial anaemia is produced in the isolated vascular region, which is followed, owing to the interrupted arterial blood-pressure, by a backward venous flow of blood, accompanied by serous and sanguineous infiltration into the walls and lumen of the intestine, which directly produces paralysis of the bowel. But this paralysis, in its turn, causes stagnation of the intestinal contents, with abnormal fermentation, and excessive production of gas. It may also cause volvulus and invagination of the bowel, as the healthy and active intestines twist themselves about the paralysed and passive portion, or project themselves into it. As volvulus of the left part of the double colon is so frequent a result of thrombosis and embolism, and as this can only be presumed to arise from active movement of this part, which must be first caused by disturbances of the circulation, we are forced to the conclusion that arterial anaemia causes at first a stimulated movement, and thereby conditions this displacement. At least we do not think that in this case the volvulus of the left portion can be explained by the intensified activity of the neighbouring healthy intestinal parts.

Finally, paralysis of the bowel leads to inflammation of the same, and to rupture, both of bowels, stomach and diaphragm, as the anti-peristaltic intestinal movement, set up in the disabled part, fills the stomach to bursting point with food matter, and the bowels, being distended with gas, either burst themselves, or by their pressure cause rupture of the diaphragm.

**Anatomic Relations of the Anterior Mesenteric Artery.**—These are very important for the due understanding of the origin of thrombo-embolic colic. As is known, this artery springs as a short strong stem, about  $1\frac{1}{2}$  inches long, from the posterior aorta. Immediately above its source the anterior mesenteric artery throws off—1st, the

superior colic artery, in a forward direction; 2nd, backwards and directly opposite the exit of the foregoing, the 17 to 20 small intestinal arteries. From this point onwards the somewhat narrowed trunk of the anterior mesenteric artery bears the name of the ileo-caecal artery. This trunk is usually the seat of worm-aneurism. It divides next into four branches—the front one, which is strongest and forms to some extent the continuation of the artery itself, is the inferior colic artery. Then follow one after the other the upper and lower caecal arteries, and finally the artery to the ileum and cæcum.

As a rule, the ileo-caecal artery is not completely blocked by the aneurismal thrombus, so that the terminal arterial branches continue to be fully supplied with blood, and no severe colic can ensue. Only when the ileo-caecal artery is entirely closed (which rarely happens) is the cæcum deprived of blood. In the case of the colon, however, on which only the inferior colic artery is blocked, the upper one, which anastomoses with the lower, remains free, and by itself continues to supply the colon with blood. The occlusion, therefore, of one colic artery does not of itself suffice to produce a fatal thrombo-embolic colic. (This is important in forensic cases.) Both colic arteries are only then affected when the aneurism implicates the source of the anterior mesenteric artery, which is rarely the case. Should this happen, then embolism of the arteries of the small intestine may follow. These, however, generally escape. On the other hand, transient colic may certainly arise from sudden stoppage of one colic or caecal artery.

If portions of aneurismal thrombus become detached, emboli may occur, but generally only in the terminal arterial branches, especially in the inferior colic artery. This one-sided embolus does not produce fatal colic, because the upper colic artery remains patent. Neither does embolus of one of the two arteries of the cæcum end fatally. For they, too, are connected, and the gut is moreover nourished by a small vessel which reaches it from the inferior colic artery.

The arteries of the small intestine finally separate near the bowel into two curved branches, each of which flows into the neighbouring anterior or posterior branch. From these curves straight channels run to the bowel. If, therefore, one embolus fix itself above the bifurcation of an arterial branch, the flow of blood is not thereby prevented, because the vascular curves and the branches springing therefrom are mutually connected.

**Symptoms and Course.**—The course of thrombo-embolic colic may vary greatly.

1. *Acute Colic Ending in Recovery.*—This not uncommon form is characterised by frequent and recurrent attacks, without any apparent cause, and which bear much resemblance to rheumatic colic, being sometimes extremely painful, but generally brief. Probably they arise from partial embolisms of the intestinal vessels (colic arteries), which are gradually worked off by anastomosis. As positive diagnosis is naturally difficult, they are often mistaken for other forms of colic.

2. *Acute Course with Unfavourable Ending.*—This refers to

all cases in which thrombosis or embolism produces displacement of the intestine. It is here also characteristic that the manifestations occur without any outwardly discoverable cause. But in contrast to other kinds of colic, the pains are often quite trivial. This is perhaps to be explained by paralysis of the local nerves by complete stoppage of circulation. In other points the symptoms agree with those of constipation colic caused by internal displacement, that is to say, they are neither characteristic nor constant. Death follows either from rupture of stomach or bowels, or else from sepsis.

*3. Chronic Course with Relatively Favourable Ending. Chronic Embolic Bowel-Catarrh.*—Consequent upon occlusion of intestinal arteries, hyperæmia, due to congestion, is first developed. Should this, owing to its situation, not lead to serious changes, the hyperæmia may last a long time, and lead eventually to chronic intestinal catarrh, as, for instance, has been noticed with congestion in the region of the mesenteric vein (portal vein) after liver disease. ("Emolic intestinal catarrh.") The course of the complaint is then generally as follows:—After relief of the first attack, the increase of temperature and pulsation disappears, but the peristaltic action continues to be retarded, evacuation is suppressed, less food consumed, and in general the condition is unsatisfactory. After one or two days low fever reappears, with intermittent, though slight, symptoms of colic, which, however, may be totally absent. In this long-drawn-out fashion it may continue for several days and even weeks (chronic, creeping colic); in one case it lasted twenty-two days. Occasionally this chronic intestinal catarrh passes into chronic emaciation and cachexia. It ends more frequently in death with the appearance of sepsis.

*4. Chronic Course with Unfavourable Ending. Development of Thrombo-Emolic, Hæmorrhagic Inflammation and Necrosis of the Intestinal Mucous Membrane. Hæmorrhagic Infarction of the Bowels.*—The pathogenesis of this hæmorrhagic inflammation may be explained by thrombo-embolic occlusion of small intestinal arteries, in conjunction with a local worm-aneurism, whereby only smaller portions of the mucous membrane of the intestinal canal suffer from the interrupted circulation mentioned before (arterial anaemia, vascular engorgement, hæmorrhagic congestion, necrosis). The chief symptoms are a greatly diminished appetite, which indeed may be altogether lost for days, increased thirst and retarded evacuation, with distinctly audible peristalsis. The faeces are at first small

pellets; later slimy, foetid and pulpy, and often bloody (blood-colic); the urine is very acid and contains phosphates and albumen; intense and lasting fever ( $105^{\circ}$  F. and more) shows itself; the pulse grows weak, but very rapid (60-70, and later as high as 90-100 per minute). The weakness increases, accompanied by wasting, a falling in of the hind-quarters, alternate dung-balls and pulpy faeces, discoloured urine and affection of the sensorium. Sometimes colic recurs immediately after eating, and the condition grows worse. Sometimes towards the fatal end remarkable paroxysms of fever are noted, viz.: violent and general quivering of the muscles and shivering; the extremities become icy cold, and the mucous membranes, where visible, turn pale; the breathing is difficult, rapid and rattling; palpitation of the heart sets in, and the internal temperature rises. The duration is generally lengthy. Often it is preceded by a prolonged chronic colic of the intestines. The actual haemorrhagic inflammation may last for days and even weeks. Death results finally from absorption of septic matter from the bowel into the blood-vessels; from peritonitis or chronic marasmus. Recovery is rare, but results from detachment of the necrotic scab and healing of the lesion; but chronic ulcers may remain behind as a consequence. In any case the stage of convalescence is very prolonged.

**Anatomical Notes.**—Dissection reveals very characteristic changes in haemorrhagic congestion of the bowels. Besides the usual signs of sepsis in the stomach, and along with swelling, injection and ecchymosis of the mucous membrane, one finds small, reddish-black blood coagula attached to plugs (thrombi) of small eroded vessels of the mucous membrane and submucosa; in the small intestine is a thin, blood-coloured fluid, with dull brown-red infiltration of the mucous membrane; the same also in the large intestine, whose walls in circumscribed places (sometimes at its junction with the mesentery) are swollen by a serous-haemorrhagic infiltration to thrice their normal thickness, or even more. Very seldom are embolic changes found in the rectum. At the spots where this haemorrhagic congestion is greatest the mucous membrane is friable and covered with a necrotic scab, strongly infiltrated with blood round its edge. At the base of this scab one can very often find, on more careful investigation, a thrombotic intestinal artery and follow it up centrally, whereby the existence of a worm-aneurism of the anterior mesenteric artery may be demon-

strated. In addition gelatinous infiltrations may be found in various parts of the abdominal cavity (especially the anterior root of the mesentery), and large quantities of bloody serous or ichorous fluid in the peritoneal cavity.

**Treatment.**—The treatment of thrombo-embolic colic is symptomatic. It consists in attempting to remove the morbid conditions arising from the intestinal paralysis, as the locality of the vascular changes in question is not accessible. As these consist chiefly in an accumulation of dung, which, if too long neglected, produces local inflammation and necrosis of the mucous membrane, it is first of all needful to remove this coprostasis by the use of purgatives, such as calomel or neutral salts. Whether physostigmine be also applicable is doubtful, owing to the fact that the intense bowel-contraction which it produces may easily cause the affected parts of the bowels to be twisted or displaced by the unaffected intestines. Diligent massage is also advisable, and gentle exercise of the sick animal, so as to remedy any stoppage of circulation, together with constant cleansing of the rectum by hand and by injection of water. The treatment of thrombo-embolic, haemorrhagic *gastro-enteritis* is identical with that of inflammation of the stomach (which see). The *dietetic supervision* of the sick animal, especially when convalescent, is of great importance. To prevent necrotic lesions of the mucous membrane calomel may be used, and various antipyretics are useful against septic fever. But, on the other hand, all drastic purgatives, such as tartar emetic, aloes or croton oil, must be avoided.

The Bollinger theory of the relation between colic and worm-aneurism of the anterior mesenteric artery has met with enthusiastic support from some and emphatic contradiction from others. Especially has Gerlach, in his "Forensic Veterinary Science," 1872, p. 531, declared himself strongly against there being any connection between the two. Röll ("Textbook of Pathology and Therapeutics") would admit within certain limits the extension postulated by Bollinger of thrombo-embolic colic to all sudden cases of colic which cannot be traced to distinctly provable causes. We ourselves are of opinion that the existence of thrombo-embolic colic has been clearly shown, and that it is by no means rare. But we hold that Bollinger certainly went too far when he declared that 50 per cent. of all fatal cases of colic are thrombo-embolic, and that every sudden case not traceable to any clear cause may be so classified.

#### CONSTIPATION-COLIC IN HORSES.

**SUB-VARIETIES.**—As many causes tend to produce constipation, so there are varied forms of this disease to be differentiated,

which, however, cannot be clinically separated. These varieties of constipation-colic, which are only etiologically different, may be caused by :—

1. Accumulation of food in the bowels.
2. Gastric and intestinal calculi (stone-colic).
3. Displacement of the intestines.
4. Invagination of the bowels.
5. New growths producing stenosis.
6. Stricture of the bowels.
7. Dilatation and paralysis of the bowels.

#### I. CONSTIPATION-COLIC CAUSED BY ACCUMULATION OF FOOD IN THE BOWELS.

**Etiology.**—The causes of this complaint have been already cited in our general remarks. They are generally to be traced to some unusual form of food. Especially dangerous are an excess of bran, straw cut too short, or an overdose of straw (straw-colic), and particularly pea and rye straw if chopped too fine, tough-fibred clover, spelt-husks, beech-nut shells, bean-stalks, in a word, any indigestible cellulose horse-feed; furthermore, all kinds of relaxing food, and a too sudden change from pasturage to stable food, or exclusive feeding with maize. Insufficient exercise combined with full feeding, as well as defective mastication arising from defects of the teeth, are both causes of feculence. Foals sometimes during the first few days after birth show signs of colic arising from an accretion of meconium in the rectum.

**Anatomical Conditions.**—The local changes produced by the accumulated faecal matter in the obstructed gut are very diverse. First of all, the substances which have lodged there dry up more and more by absorption of their moisture into the bowels, and thus become sources of irritation to the surrounding membrane. This causes, in the first place, catarrhal inflammation of the mucous membrane, with profuse ecchymosis (haemorrhoidal catarrh), and, later, diphtheritic and necrotic conditions. According to Ernst, the diphtheritic inflammation of the mucous membrane begins with small spots, at first grey and afterwards greyish yellow, or even white, which show especially on the projecting portions, and make its surface look as though sprinkled with bran. These spots are caused by a superficial necrosis of the membrane, which

leads to the formation of ulcers and pustules ; only in rare cases is there any deeper necrotic injury of the tissue resulting in perforation. A further consequence of faecal stasis is rupture of the stomach and bowels. Thus faecal stasis in the cæcum (unless removed) appears regularly to produce rupture of the affected bowel, and in the colon and rectum very frequently. Faecal stasis in the passage from the ileum to the cæcum is, in a moiety of cases, followed by rupture of the stomach, in which a so-called anti-peristaltic intestinal movement, arising in one of the obstructed parts, drives the contents of the bowel back upon the stomach, overfills it, and causes it finally to burst, unless it be relieved by vomiting. Food accumulations occur most frequently in the ileum, just before its point of discharge into the cæcum.

**Symptoms.**—The manifestations of colic produced by accumulation are less marked than in other forms. The pains are usually mild, the animals generally lie still, and groan occasionally. They cease to take food. The passage of faeces is delayed, the peristaltic movement is suppressed, the separate dung-balls are very dry, small and often covered with slimy shreds. On examining the rectum, during which the animal generally presses and strains violently, the gut will be found full of solid faeces. As the overcharged gut presses upon the bladder, the animal makes frequent attempts to urinate, whereby its abdomen becomes gradually fuller of gas and is at last distended. The disease runs sometimes for a considerable time as a simple constipation without any colic symptoms (as with dogs); but if it last very long, the latter signs begin to show themselves. The complaint is always of prolonged duration, often very much so. Usually it lasts several days, very frequently two or three weeks, and in some cases six or seven weeks ("chronic" colic). Meanwhile there are no true remissions, such as are characteristic of thromboembolic colic. Vomiting is rare. The symptoms of bowel rupture, which is too often fatal, closely resemble those described under the head of stomach rupture.

**Therapeutics.**—The treatment consists in the administration of laxatives, also in massage of the abdomen, cold bandages, liberal injections of water, moderate exercise and low diet. Dose at first with Glauber salt, or artificial Carlsbad salts in  $\frac{1}{2}$  to 1 lb. doses. By some the use of castor-oil in combination

with other oils is much recommended. They give for several consecutive days a mixture of 1½ pints of poppy-seed oil, or olive-oil, with 8–16 oz. of castor-oil. Also eserine (½–1 grs.), in conjunction with pilocarpine (4 grs.). Arecoline (1 gr.), as well as aloes (8–12 dr.), are often used with good results. Eserine seems, however, to be more suitable for milder cases of the complaint; in more serious cases it should not be used at all, or only with great caution, owing to the risk of bowel-rupture. Against tympanites, which sometimes ensues during constipation-colic, Albrecht recommends creolin (every three hours in doses of 1 dram, combined with laxatives).

**2.—CONSTIPATION-COLIC, FOLLOWING UPON GASTRIC AND INTESTINAL CALCULI (stone-colic) and other foreign substances in the intestinal canal (sand-colic).**

**Occurrence.**—Stone-colic is, as a rule, rare. Among 1,882 fatal cases of colic in the Prussian Army (1889–93) only 25, or 1·5 per cent., were authenticated as stone-colic. It occurs most frequently among the horses of millers and bakers, which are almost exclusively fed upon bran and flour refuse. Bran, for instance, contains 1 to 2½ per cent. of phosphate of magnesia, which is the chief ingredient of these calculi. The small proportion of millstone dust, which is mixed with the bran, seems to be of less account. The so-called sand-colic must also be mentioned here. It arises from swallowing sand or minute stones, when drinking from shallow brooks or pools; from eating muddy or sandy fodder, on the way to pasture, or after the stable gangway has been sanded. Finally, the animal's habit of licking itself when shedding its coat tends to the formation of balls of hair and consequent stoppage. Other foreign bodies, such as bits of cloth, needles, etc., when swallowed, are often causes of constipation.

Intestinal calculi, or enteroliths, consist for the most part of ammonio-phosphate of magnesia (90 per cent., Fürstenberg), also of phosphate and sulphate of calcium, phosphate of magnesia, silicates, chlorine compounds, traces of iron, as well as of organic substances (mucus, epithelium and particles of fodder). Their formation is explained by the combination of the phosphate of magnesia, so largely present in bran, with ammonia compounds present in the intestines, and which reach them from the ammonia-laden air of the stable through the drinking water consumed, and afterwards combine to form

an insoluble compound. The core of the stone is usually some foreign substance, a grain of sand, or of oats, or the head of a nail, around which the accretion of phosphate is formed. So-called stomach-calculi are nothing else but bowel-calculi, which, by the anti-peristaltic movement of the bowels, have been forced back into the stomach; for food remains so short a time in the latter that no calculus can be formed there in presence of its strongly acid contents. The formation of calculi usually occurs in the large intestine, and particularly in the greater curvature of the colon—much more rarely in the caecum. Colin found, for instance, in 900 dissections, that the stone was in the colon in 23 cases, and only once in the cæcum. In addition to calculi, other accretions may lead to constipation, such as collections of sand or mud mixed with vegetable fibre, mucus, hair, etc., and there are so-called false calculi, which are such accretions coated over with phosphate. The local changes produced by bowel-stones are the same as those described in connection with faecal stasis. In sand-colic large quantities of sand are often found in the intestines—30 to 50 lbs., and even more.

**Symptoms.**—The symptoms of constipation-colic produced by calculi are often not to be distinguished from those of a stoppage or accumulation colic. Frequently, prior to the stone becoming wedged in the bowel, the animal has never been ill, in spite of its presence there. In other cases frequent colic has taken place, and sometimes at regular intervals. In general, the attacks are more severe in cases of stone-colic than of stoppage-colic, and the constipation is more obstinate. Then vomiting and rupture of the stomach and bowels are more common in stone-colic. Its course is generally shorter, though it may at times be prolonged. The prognosis is much more unfavourable.

A positive diagnosis of stone or sand-colic can only be made when bowel-stones or sand are evacuated through the rectum, or after examination with the hand. Only with millers' horses, particularly if frequently affected, may the presumption be ventured upon that they are suffering from stone-colic, though other forms of colic, especially that which proceeds from over-feeding, are not uncommon with them.

**Therapeutics.**—The treatment is the same as for colic caused by accumulation. Eserine must, however, in all cases of

stone-colic be avoided, owing to the risk of intestinal rupture, but in sand-colic it may, on the contrary, be with advantage administered in small doses. In desperate cases, the firmly wedged stone may be removed by operation, if it can be located. Thus Félixet removed a stone the size of a child's head by flank incision. On the other hand, horses operated on by Hall, Rickards and Dollar died after 24 to 27 hours. The proposal of Trasbôt to drive back the stone by generating carbonic-acid gas in the rectum by means of soda and tartaric acid could scarcely be carried into practice. French veterinary surgeons recommend for sand-colic in military horses the drinking of immense quantities of water—say 20 to 25 gallons daily, or in 8 days, about 160 to 200 gallons.

### 3.—CONSTIPATION-COLIC DUE TO DISPLACEMENT OF THE INTESTINES.

**Anatomical Notes.**—The internal changes of position of the intestine consist of volvulus and twisting or incarceration of the intestinal parts. Volvulus most frequently affects the colon (more particularly its left layers) and the small intestine. Volvulus of the cæcum is rather a kinking, with displacement of the point downwards and backwards, and a shifting of the base upwards and forwards. Volvulus of the rectum is rarer. Twisting or knotting arises from the coiling of the small intestine in loops around some portion of itself, or of the colon and rectum, whereby they are twisted round the mesentery. Or it is caused by omental cords wrapping around loops of the small intestine, and less frequently by the cæcum enveloping the colon. Incarcerations, finally, occur specially in congenital or acquired openings in the abdominal cavity, thus in the inguinal ring, in Winslow's foramen, in old fissures of the diaphragm, in apertures of the omentum and mesentery, in clefts formed by ribbon-like adhesions, the detached ureter, or adhesions of the omentum to the scar left by castration.

**Etiology.**—The real causes of these internal displacements are for the most part obscure. Some certainly belong to the sphere of thrombo-embolic colic, and may be explained as already stated. In other cases they arise from an unequal filling of certain bowel-loops with gas or with very heavy food, whereby these loops are constrained by gravitation to seek a change of position. Moreover, acute peristalsis, such as

occurs in severe colic, appears often to produce displacement, though only in the later stages of the colic. It is therefore not impossible, as many observers have supposed, that the use of eserine frequently produces displacement. Finally, the contortions and violent plunging of the animal are, in rare cases, a cause, although this origin is much too frequently assumed. The drinking of cold water, once so largely blamed, can scarcely be seriously considered as a cause.

**Symptoms.**—The phenomena have nothing characteristic about them. Persistent vomiting, lasting several days, is a symptom which may just as often be absent as not. And the strange attitudes and signs of pain, movement to and fro of the head, lying on the back, the appearance of stomach or bowel rupture and absence of faeces, all occur in other forms of colic arising from constipation. The duration varies greatly, according to the completeness or otherwise of the bowel stoppage. In the latter case it may last eight days, but in complete stoppages death usually occurs within a day. Often the colic signs are of a transient character, and return periodically, when, for instance, a bowel-loop is alternately temporarily imprisoned and released. Possibly the brief attacks of colic of so-called "night kickers" may be so explained.

A diagnosis of constipation-colic produced by internal displacement is only possible in rare instances, and then only when an incarcerated inguinal hernia, or some other incarceration or twist, can be demonstrated from the rectum. Torsion of the colon and cæcum can sometimes be verified in this way. Finally, the fruitlessness of the treatment adopted may also guide in forming a diagnosis.

**Therapeutics.**—As a rule, treatment of internal displacement is not successful, excepting in case of incarcerated inguinal, umbilical or abdominal hernia, which may be reduced by taxis, or by incision of the sac, enlargement of the hernial orifice and replacement of the constricted bowel-loop, if it be not already necrotic. In addition to the use of purgatives and enemas, attempts may also be made to detach from the rectum any connective tissue which may be adhering there, if such be the cause of the constipation; or the sick animal may be moved into various positions, rolled over, led uphill and downhill—though usually without effect. The same may also be said of the former practice of administering large doses of mercury. In fact,

the only remedy for many internal displacements, viz. :—laparotomy and manual rectification, can usually not be adopted, owing to the uncertainty of the diagnosis and the danger of an operation.

According to Jelkmann the diagnosis of a volvulus of the left layers of the colon should be positive, if, on inserting the left hand, immediately in front of the pelvic entrance near the fourth lumbar vertebra, the posterior mesenteric cord is felt to be tightly stretched, and in a diagonal position, instead of being perpendicular. It should be exceedingly sensitive to touch. If the twisted layers of the colon be tightly packed or distended, a second tensely strung cord will be found in the left region of the flank (bands of left inferior colon layer). According to Jelkmann this volvulus may be removed as follows :—Pass the left hand towards the left abdominal wall and push the left layers of the colon forwards and inwards. On reaching the region of the median line, raise the hand slowly, whereupon the upper layer of the colon, with the lower one, will sink over the hand into its normal position. By the upward movement of the hand the twisted loops of the rectum are also said to be lifted, so that both layers of the colon are able to fall back upon the left inferior abdominal wall. These statements have been confirmed by others. The Bavarian military veterinary report of 1891 states that Jelkmann's method had been applied with partial success in Bavaria twenty-five years earlier. The operation is facilitated by slightly raising the posterior parts of the horse.

#### 4.—CONSTIPATION-COLIC CAUSED BY INVAGINATION OF THE INTESTINE.

**Definition.**—Invagination, or intussusception, consists in the intrusion of any intestinal part into the lumen of a neighbouring part. Most frequently, it is the ileum which is projected into the cæcum, and often, indeed, to a very considerable length; or it consists in an invagination of the small intestine upon itself. Less frequently the cæcum is thrust bodily into the colon. The rarest cases are invaginations of the rectum into itself. In one case upon record, the small intestine had penetrated into the cæcum as far as the colon and rectum. The causes of invagination are generally to be sought in paralysis of the peripheral (distal) parts of the intestine, and penetration into the former of the more actively peristaltic central (proximal) portions. In one case the phenomena of invagination were noticed in a saddle horse immediately after jumping.

**Symptoms.**—In cases of invagination-colic these agree with those caused by internal displacement. The duration is, however, generally longer, because, in cases of invagination, the whole lumen of the bowel is not usually blocked. A case

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is known in which, after ten days of diarrhoea and apparent improvement, death took place in 27 days. Indeed, animals with intussusception of the cæcum into the colon often live for a long time, and seem, in fact, able to accustom themselves to this condition. In rare cases, a definite recovery is made by casting off the invaginated part of the bowels, and the growing together of the two ends of the intestine.

A positive *diagnosis* of invagination can only be made when necrotic, tubular portions of intestine are passed.

The *treatment* is consequently somewhat uncertain, and can, at best, only consist in flooding the intestines to procure easier passage, in the administration of purgatives, and in the introduction of large quantities of water into the rectum.

### 5.—CONSTIPATION-COLIC CAUSED BY NEW GROWTHS.

**Anatomical Notes.**—New growths in the intestinal canal are rare—consequently do not often produce colic. They either narrow the lumen of the gut by affixing themselves to its side, or by compression from without, as, for instance, when situated on the mesentery. Thus, stomach-polypus may obstruct the duodenum and stretch it even to rupture. The lumen of the small intestine may be narrowed by fibromata, melano-sarcomata, myxo-sarcomata, leio-myomata and sub-mucous lipomata, whereas in the large intestine and rectum the new growths are chiefly sarcomatous or cancerous. Lipomata occur rather frequently on the mesentery, where they cause bowel constriction or strangulation. Finally, stenosis of the intestine may be produced by abscesses on their walls, polypus in the rectum, proliferations of the intestinal mucous membrane resembling valves or pouches, tumours in the ovaries (ovarian cysts), as well as by phlegmonous inflammation of the connective tissue of the pelvis. The contents of the stomach usually lodge in front of the constriction, and cause expansion and compensatory thickening of the walls.

**Symptoms.**—The phenomena of this rarer form of colic consist in chronic constipation with intermittent attacks of colic, which grow more and more intense. This condition may last for years, until finally, when the stenosis is more developed, the animal perishes from faecal stasis. New growths in the rectum show themselves by the straining of the animal during evacuation, and the passing of blood-stained faeces. This may be confirmed by examination of the rectum.

**Therapeutics.**—The most important point is to keep the bowel contents in a liquefied condition by the exhibition of purgatives. When the new growths can be felt per rectum they may in some instances be removed by scraping, or if pedunculated by torsion. In all cases the rectum should be emptied manually several times daily.

#### 6.—CONSTIPATION-COLIC CAUSED BY INTESTINAL STRICTURE.

**Etiology.**—Structures productive of stenosis of the intestine arise from the cicatrization of ulcers of the mucous membrane following inflammation of the bowels, and of previous imprisonments or invaginations. In this way the lumen of the bowel may be reduced to the thickness of a finger. A corresponding dilatation of the intestine is always found in front of the stenosis. Stenoses occur most frequently in the ileum.

**Symptoms.**—These closely resemble those mentioned in other forms of colic due to constipation. The duration is usually very prolonged (habitual colic), and the animals often grow very thin during the complaint, in spite of an excellent appetite.

**Therapeutics.**—Treatment is only possible in stricture of the rectum, which can be reached with the hand. This should consist in gradual expansion of the constricted part by frequent introduction of the arm. In one case Mauri successfully performed a partial resection of the narrowed part of the rectum. Other strictures, and especially the very dangerous one of the small intestine, are beyond the reach of treatment.

#### 7.—CONSTIPATION COLIC CONSEQUENT ON DILATATION AND PARESIS OF THE INTESTINES.

**Etiology.**—Enlargements of the stomach and bowels are usually the secondary results of constriction (stenosis), caused by new growths, stricture, fecal stasis, calculi or invagination, and are mostly found immediately in front of these, being complicated by severe hypertrophy of the intestinal wall. Stomach enlargement may also be caused by stenosis of the *villus*, produced by the narrowing of the intestine, *caecal* through stoppage by food. A less frequent source of such enlargement is the paralytic condition

of the intestinal wall following upon abnormal distension by food or gas, or after embolic obstruction of the intestinal arteries. Local distensions of the bowel-walls (so-called diverticula) are mostly the result of previous partial ruptures. How far chronic stomach and bowel catarrh give rise to enlargements by their interference with the due nourishment of their respective walls, it is difficult to decide with the material at present before us.

**Symptoms.**—Practically the most important of these are the enlargements and pareses of the wall which occur in the rectum. They induce chronic, lasting and often intermittent colics (habitual colic), which end in rupture of the intestine. On examination, the rectum is found crammed with truly enormous masses of dung, on removal of which the colic symptoms vanish. These faecal masses may even exert pressure upon the pelvic portion of the urethra, producing stoppage of urine in the bladder, from which hypertrophy and dilatation of the bladder may result. In severe cases of stomach distention, the pressure of this organ upon the diaphragm may cause extreme dyspneic symptoms during the course of the colic. Leisering describes a case in which the circumference from the cardia to the pylorus on the greater curvature amounted to 50 inches, and on the smaller to 36 inches.

**Treatment** is the same as with constipation colic produced by bowel stricture. In enlargement and paralysis of the rectum the treatment consists mainly in emptying it by hand. This must be frequently and regularly done so as to prevent renewed attacks of colic. A radical cure is impossible.

#### 8.—FLATULENT COLIC.

**Etiology.**—This form of colic is usually caused by the consumption of food tending to produce flatulence. The foodstuffs specially provocative of fermentation have been already mentioned in our remarks on the general causes of colic. Moreover, windsuckers when they swallow too much air are sometimes attacked by this form of colic, and according to some authorities, horses may occasionally suffer from it after moving rapidly against the wind. The gases developed in flatulent colic are generally carburetted hydrogen and carbonic acid. Pinner found 49 per cent. of carburetted hydrogen, 8

per cent. carbonic acid, and 42 per cent. of nitrogen. Hydrogen and sulphuretted hydrogen are also present.

**Symptoms.**—The phenomena of flatulent colic consist in a very rapidly developing distension of the abdomen by the above-named gases. The abdominal walls are then arched like a barrel, are very tense to the touch, and, on being struck, the flanks emit a loud and clear sound, sometimes even a metallic and re-echoing ring. On applying the ear very loud tinkling noises are heard. As this inflation increases, so also does difficulty of breathing, and the animal becomes anxious and restless. The visible mucous membranes are much stained with red, sometimes bluish, the jugular veins are prominent, and the heart palpitates. The animal's gait becomes stiff, and it staggers in the hinder parts. Its recovery is shown by abundant discharge of intestinal gases, and by the sinking of the abdomen to normal shape and size. A fatal result is caused sometimes by compression and oedema of the lungs; at others, by blood poisoning with carbonic acid, or by apoplexy, and finally by rupture of the bowels or diaphragm. The course of the disease is always very rapid.

Flatulent colic is not difficult to *diagnose*. The gases which accumulate during other forms of colic (thrombo-embolic colic and that due to constipation) may be known by their slower and more gradual appearance and their longer persistence; for flatulent colic is always rapid in its development.

**Therapeutics.**—In the treatment of wind-colic, the most important matter is the puncture of the intestine at the right moment, as this is the only certain method of removing the accumulated gases. It is usually made in the region of the right flank, in the centre of a triangle determined by the external angle of the ilium, the posterior edge of the last rib, and the ends of the costal processes of the lumbar vertebrae. First of all the skin and trochar must be disinfected, hair clipped off, and the perforation then made with a lancet or fleam, the point of the trochar being directed downwards and forwards towards the left elbow. Then the wound should be closed with collodium or sticking plaster. But if the left flank happens to be more inflated, or the only one accessible, the puncture may be made there instead. Puncture from the rectum, as already suggested, has lately been strongly recommended by some.

In many cases of slight tympanites, physostigmine ( $\frac{1}{2}$ — $1\frac{1}{2}$  grs.), and arecoline ( $\frac{1}{2}$ — $\frac{1}{4}$  grs.) do good service. But where the bowels are very tightly stretched, both these remedies are not without danger, owing to the risk of bowel-rupture. Enemas of ether, as used largely in France (viz. : 4 to 6 drams of ether in one quart of water), are also sometimes useful in slighter cases of wind and cramp colic. Other remedies used against flatulent colic are of much less value.

Care must meanwhile be taken to assist the passage of flatus by cold, as well as stimulating enemas; cold cloths and kneading of the abdomen. The administration of so-called "absorbent remedies" is not to be commended. Some of them, such as spirits of ammonia and sulphuretted potash, actually increase the amount of gas by the addition of ammonia and sulphuretted hydrogen; while others—lime-water for instance—are scarcely able to retain their neutralising properties beyond the stomach.

#### 9.—COLIC DUE TO WORMS.

**Etiology.**—Among the parasites which may produce colic the first in order of importance is *Ascaris megalocephala*, then *Tænia plicata*, *mamillana*, and *perfoliata*, *Spiroptera megastoma*, *Strongylus armatus*, *Scelerostomum tetracanthum*, and finally *Oxyuris mastigodes*, as well as *Gastrophilus equi* and *haemorrhoidalis*. They may cause colic in two ways—either by forming knotted balls which cause obstruction (this occurs with both round and tape-worms), or through traumatic injuries, which provoke inflammation, ulceration, and even perforation of the walls. In the latter connection, the *gastrophilus* must be specially mentioned, as, although generally innocuous, it may, in isolated cases, cause fatal colic. *Ascarides* may also produce multiple ulcers and violent inflammation of the intestinal mucous membrane, if present in very large numbers. Perforation of the intestine, with subsequent suppurative peritonitis, has also been found caused by *Tænia perfoliata*. Moreover it must not be forgotten that intestinal worms, and especially nematodes, are often found on dissection, and care must be taken not at once to connect their presence etiologically with a preceding colic.

**Symptoms.**—No special phenomena attend worm colic. Young animals are most liable to be affected, owing

to their lesser power of resistance to the parasites. The colic is usually recurrent, but of slight intensity; but with excitable mares, extraordinarily violent and frantic attacks of colic pain may arise, owing to the presence of *gastrophilus haemorrhoidalis* in the rectum. The course of the complaint is generally chronic, in which digestive disturbances alternate with attacks of colic; but it may become acute if perforation of the stomach walls occur, or absolute stoppage of the lumen of the bowel, or in cases of intestinal inflammation. In these last named forms it is more fatal; otherwise the prognosis in worm colic is not so unfavourable.

**Therapeutics.**—These consist in the administration of anthelmintics. First of these is tartar emetic. It is given dissolved in drinking water, say 2 to 4 drs. per diem. Other remedies are—arsenic, powdered in single doses of 15-30 grains; turpentine, 2 to 4 oz. in emulsion; santonine (2 to 6 drs.) and areca nut (3 oz.). For the larvæ of *gastrophilus* in the rectum we recommend enemas of soapy water, as well as dilute solutions of petroleum, benzine or creosote, together with manual removal.

#### COLIC IN CATTLE.

##### I.—COLIC IN CATTLE CAUSED BY INVAGINATION OF THE BOWELS.

**Etiology and Symptoms.**—This is certainly the most frequent cause of colic in cattle. Indeed, it seems to occur oftener in cattle than in horses. The origin of intestinal invagination is supposed to lie in a previous chill, severe exertion in climbing a hill, and the more active peristalsis thereby produced. The consequences are very characteristic. The animals are suddenly seized, without any previous indigestion, by a more or less severe colic, which lasts six to twelve hours, and then completely disappears, so that it seems as though the patient had entirely recovered. But loss of appetite, suppressed rumination and the passage of faeces, which are at first diarrhoeic and later dry, all point to a more deeply seated malady. Soon persistent and very obstinate constipation, which defies all purgatives, sets in, during which, and only after much straining, small, tough, slimy lumps of fibrous masses, sometimes smeared with tar-like blood, are evacuated. The peristaltic sounds are at the same time quite suppressed, the abdomen is gradually

inflated by the generation of gas in the intestines, and is sometimes sensitive to pressure over a certain area; the pulse becomes feeble and very quick, while the temperature is out of all relation to this rapid pulse, the body feeling cold to the touch. The beast stands in dull apathy. Retching is noticed in isolated cases. On examining the rectum, one may sometimes find in the course of the bowel a longish, cylindrical and painful swelling.

**Course.**—This is rather slow. Generally the animals die, often without any death struggle, on the sixth to the ninth day, sometimes after about fourteen days, but seldom as early as the fourth. Occasionally the attacks recur several times at intervals of eight to twelve days. Recovery is very rare, and happens then through necrotic amputation of the invaginated part, with peritonitic adhesion and growing together of the severed ends of the intestine. Tubular pieces of intestine have in such cases been found in the faeces.

**Diagnosis.**—It is generally not difficult to recognise invagination of the bowels in cattle. Sudden attacks of colic followed by unusual quietness, very obstinate constipation, evacuation (with much straining) of bloody slime, absence of the sounds of digestion, severe general derangement, local sensitiveness of abdomen (especially on the right), the growth of a painful swelling on the course of the intestine, and the duration of these conditions for several days, with gradual increase of debility, are all points to aid in diagnosis. When exploring the rectum care must be taken not to mistake the left kidney for an abnormal tumour, as it normally projects some distance into the abdominal cavity (Gautier).

**Therapeutics.**—The treatment is chiefly surgical. The administration of purgatives generally does no good, so that no time should be wasted in giving them. Attempts to loosen the intussusception by simple physical means, by repeatedly driving the sick beast up and down hill, seldom succeed. The operation consists in opening the abdominal cavity at the right flank, seeking for the invaginated spot (very difficult in cases of pregnancy), and separation by hand of the invaginated parts. If this does not succeed, then the invaginated portion of bowel must be cut off, and the ends joined by Lembert's sutures.

**2.—COLIC IN CATTLE CAUSED BY STRANGULATION OF THE INTESTINES.**

**Etiology.**—Apart from the occasional strangulation of the bowels in perforations of the omentum, mesentery, diaphragm, broad uterine ligament, etc., bowel incarcerations are most commonly met with in cattle in the so-called pelvic hernia or gut-tie. As is well known, this is traced to a peculiar method of castration, namely, traction on the spermatic cords. Injurious results from this method may follow in three ways—either a rent is formed in the peritoneal fold of the spermatic cord which runs along the edge of the pelvic entrance, into which an intestinal loop insinuates itself from before backwards, and becomes strangulated. Or by the sudden rupture of the spermatic cord the proximal end is jerked backwards into the abdominal cavity, and there twists round the intestines, producing the so-called entanglement. In either case an imprisonment of intestines is caused. Less frequently the stump of the spermatic cord adheres to the wall of the abdomen, and the cleft thereby formed is sometimes a cause of imprisonment.

**Symptoms.**—These are in general almost the same as those described in invagination of the bowels. The duration is also about the same, being six to eight days. For diagnosis the discovery of a doughy and painful swelling, in size between that of an apple and the fist, situated on the edge of the pelvic orifice, a hand-breadth from the internal right abdominal ring, is of first importance. The spermatic cord will also be found tensely strung.

**Treatment.**—This is purely surgical. The imprisoned bowel is either freed by taxis through the rectum, pressing the loop forwards and downwards, and pulling the tightened spermatic cord from its position; or an incision is made in the flank, and the imprisoned part set free by the inserted hand.

**3.—OTHER FORMS OF COLIC IN CATTLE.**

Colic rarely occurs in cattle, except from the two causes named, and from inflammation of the bowels. (See chapter on Gastro-Intestinal Inflammation.) As with horses, it may be developed by over-feeding, chill, stoppage of the bowels, new growths, faecal matter, hair-balls, the swallowing of after-

birth, volvulus, stenosis of the intestine by congenital semi-circular valves or new growths (lipomata), prolapse of the liver through clefts of the diaphragm, etc. But the attacks are generally not so severe; only rarely are plunging, rolling on the back, bellowing or leaping into the manger to be seen. The animal is generally restless, stamps, strikes its foot against the belly, lashes its tail, and frequently looks backward. The treatment consists, as with horses, in the use of physostigmine ( $1\frac{1}{2}$  grs.), and of laxatives (sodium sulphate, 1 to 2 lbs.; aloes, 1 to 2 oz.; castor oil, 1 pint mixed with rape-seed oil or poppy-seed oil, 1 quart; croton oil, 15-25 drops), together with enemas and external friction.

**Constipation.**—Simple constipation unaccompanied by colic may result from the same causes as have been indicated for colic (over-feeding, improper food; stenosis of the bowels, and especially of the rectum, by pelvic abscesses and new growths), and also by coprostasis, etc.

## COLIC IN DOGS.

**Etiology and Symptoms.**—Serious colic very rarely occurs in dogs. (Among 70,000 sick dogs treated in the Berlin dog-hospital during nine years, only three cases were really severe colic.) But, on the other hand, we not unfrequently observe symptoms of restlessness, proceeding from pain in the abdominal canal, and to which the name of colic might be given. The causes are: cold, accumulations of hard food, especially of bones, obstruction of the bowels by stones and other foreign bodies, intestinal parasites, and especially invagination of the bowels. But stress must be laid upon the fact that, although these things *may* cause colic they do not necessarily do so, and that genuine colic phenomena are so slight that one rarely hears of colic among dogs. Usually obstruction of the bowels by foreign substances and faecal matter, bowel invagination as well as helminthiasis, run their course without any colic. The most frequent form relatively is one caused by worms (*Taenia echinococcus*), in which, according to Roell, even maniacal attacks may arise. The colic symptoms consist in excitement, whining, restless running to and fro, frequent lying down and curling up. They seldom last long. The pains of muscular rheumatism are sometimes mistaken for colic in the dog.

Treatment will depend on the cause. The following are given

for the purpose of alleviating pain :—Morphia-injections ( $\frac{1}{2}$  to  $1\frac{1}{2}$  grs.) ; opium in the form of Dover's powders (7 to 30 grs.) ; or tincture of opium (20-40 drops) with mucilage. Enemas may also be given, and hydropathic wrappings placed round the abdomen. Foreign substances in the bowels, volvulus of the stomach, and bowel-invagination can only be satisfactorily dealt with by operation.

#### COLIC IN SWINE.

**Etiology and Symptoms.**—Colic in swine is generally traceable to overfeeding with bad and indigestible food, bran, fermenting refuse, beer-grains and to the eating of sand (sand-hunger). Cold also and the presence of intestinal worms (*Ascaris lumbricoides* and *Echinorhynchus gigas*) may produce colic. Finally, every inflammation of stomach or bowels—such as poisoning or invagination of the bowels—produces colicky pains. The animals show great unrest, arch their backs, lie down, frequently groan and scream, stretch themselves on the ground and manifest muscular contortions and cramp. They also cease to take food, and evacuation is retarded.

**Treatment.**—This is restricted to the administration of purgatives, anodyne electuaries, enemas, friction of the belly, etc. At the same time, warm shelter and soft litter must be provided. Calomel ( $\frac{1}{2}$  to 1 dr.) is recommended as an aperient.

#### ULCERATION OF THE STOMACH AND INTESTINES.

**General Notes.**—This is seldom seen as an independent disease in domestic animals. It occurs chiefly as a secondary phenomenon in the course of other diseases, such as rabies, rinderpest, malignant catarrhal fever, petechial fever in horses, dysentery, gastro-enteritis, gastro-intestinal catarrh, poisoning by corrosive substances, or injury caused by swallowing foreign bodies, and the larvae of *Gastrophilus*, *Spiroptera sanguinolenta*, etc. These ulcers may, however, under certain circumstances, produce a definite aspect of disease. There are two kinds to be considered : 1st, ulcers arising from inflammation, particularly the so-called catarrhal ulcers and haemorrhagic erosions ; and, 2nd, the so-called peptic, round ulcers of the stomach and duodenum.

**Etiology and Pathogenesis.—**I. *Ulcers of inflammatory origin* develop after inflammation of the mucous membrane of the stomach and intestine, when the inflammatory infiltration has been very severe or accompanied by excessive haemorrhage into the tissue of the mucous membrane. In such cases, even after the inflammation has abated, permanent ulcerous losses of substance may accrue to the membrane. These losses occur at various points in the stomach, but in the intestine they are usually found on the lymph follicles (follicular ulcers). Most frequently they form so-called catarrhal ulcers in the course of acute and chronic gastro-intestinal catarrh. Even after injury and partial cicatrization of the mucous membrane, ulcers may remain behind in any part of the intestinal canal.

2. *The so-called round peptic ulcer* occurs in the stomach and duodenum only as far as the acid gastric juice reaches. The opinion used to be held that it arose—in contradistinction to that produced by inflammation—from a local self-digestion of the stomach. The sources of this gastro-malacia were sought either in local disturbance of the circulation, which hindered the regular neutralisation of the acid gastric juices by the alkaline blood, whereby the mucous membrane was exposed to its acid action, limited stoppage of the vessels of the mucous membrane by thrombi and emboli; fatty, sclerotic and amyloid degeneration of the vessels; local vascular spasms. Or they were found in an abnormal acidity of the gastric juice, which changed the slightest abrasions of the mucous membrane, which would under ordinary conditions speedily heal, into ulcers. General anaemia of the body, as well as venous hyperæmia (due to over-charging) of the mucous membrane were supposed to have precisely the same effect. Here, too, the mucous membrane, being weakened by deficient supply of arterial blood, was believed to be unable, if injured, to withstand the action of the gastric juices. Nowadays the ulcers are believed to originate in the mucous membrane by specific wound-infection (inflammation followed by necrosis). In what manner ulcers of the stomach may be caused by general combustion, or by poisons contained in the blood, is a question which remains to be further investigated. Experiments show that they may be produced by injury of the spinal cord, of the corpora quadrigemina and of the medulla oblongata.

**Peptic Ulcers of the Abomasum in Calves.**—Of this R. Ostertag has described six cases. Most of these ulcers were perforating,

and the stomachs were all in an intensely catarrhal condition. With respect to the pathogenesis of the ulcers, examination, both macroscopic and microscopic, showed that thrombosis took no part in it, but that they arose from circulatory disturbance was clearly indicated by their form. Ostertag establishes, moreover, the connection between the stomach-catarrh and the ulcers, by calling attention to the catarrhal process combined with nutritive derangement.

**Anatomical Notes.—1.** *Catarrhal ulcers of the Stomach* usually follow severe hyperæmia of the mucous membrane; and so-called haemorrhagic erosions, catarrhal inflammations of haemorrhagic nature. These erosions are most frequently found in the right half of the stomach, near the pylorus, and form small, round, superficial defects of the mucous membrane, with sharp, smooth edges and uneven base, generally covered with blood. Catarrhal ulcers usually arise in the bowels from previous follicular ulcerations, which in their turn have been preceded by extreme cellular infiltration of the lymph follicles, with subsequent destruction of the tissue, and present the appearance of small, scattered, pan-shaped ulcers. As the disease advances they change their aspect. First, they grow larger—the size of half-a-crown or the palm of the hand; later, the ulcerous action is no longer confined to the mucous membrane, but attacks also the submucous and even the muscular coats, when bleeding from the eroded vessels of the bowels often supervenes. Lastly, even the serous covering may be attacked, which gives rise to peritonitic adhesions and perforation. Along with this progressive course a healing process is also going on. Many ulcers heal completely, leaving radiating scars. In other larger ones the edge heals over, grows thicker, and often as hard as cartilage, while the neighbouring mucous membrane arranges itself in striated folds, and the submucous and muscular layers are replaced by firm, fibrous connective tissue. Signs of chronic catarrh are also present.

**2.** *Peptic ulcers* have been hitherto but little observed. They occur only in the stomach and at the commencement of the duodenum, and are thereby distinguishable from the others. They are usually quite regular in shape, roundish or elliptical, have sharp, smooth edges, appearing as if punched out; and they generally occur singly. They heal with a radiating scar, and may produce peritonitic adhesion, as well as internal bleeding and perforation.

**Symptoms.—**It is impossible clinically to differentiate

between these two forms of ulcer; their symptoms are similar and often very characteristic. The aspect of the disease is frequently exactly that of chronic gastro-intestinal catarrh. Thus Siedamgrotzky noted in a horse only gradual wasting and exhaustion; and Grad and Zippelius in a cow the symptoms of chronic catarrh of the abomasum, viz.: chronic tympanites, constipation, anaemia, and general disturbance of nutrition. Sometimes, it is true, more striking symptoms are seen, from which the presence of an ulcer can with some probability be diagnosed. These consist in obstinate vomiting, signs of colic, haematemesis, and the passage of blood with the faeces. In horses periodic attacks of colic, recurring at certain times after meals, have been chiefly noticed, with gradual falling off in appetite and even the vomiting of blood-stained matter. In cattle Gotteswinter observed, along with anaemia and uncertain gastric disturbances, that the dung was black and tar-like, indicating the presence of ulcers in stomach or bowels, which was confirmed on slaughter. Wesikopf noted several cases of rupture of the stomach in horses, caused by ulcers in the stomach. According to Rasmussen the presence of perforating ulcers in the abomasum of calves may be presumed (in life) by the animals growing restless after transport, throwing themselves down, lying languidly upon the ground, puffing and staggering and tumbling when lifted up. The course of the sickness caused by ulcers in stomach or bowels is usually very protracted. Recovery is possible, but the termination of a case which admits of positive diagnosis is usually fatal, death resulting from loss of blood, from perforative peritonitis, or, finally, from gradual exhaustion and wasting.

**Differential Diagnosis.**—Owing to the difficulty of precise diagnosis, and the usual lack of pathological indications, this complaint is often confounded with others. In particular is this the case with gastro-intestinal catarrh, which is easily understood, seeing the close connection between the two. But even when the characteristic symptoms are present, such as vomiting and haemorrhage of stomach and bowels, it is not always easy to distinguish this disease in cattle from that produced by the presence in the stomach of foreign substances, as precisely similar symptoms occur. In such a case reliance must be placed mainly upon the presence of pain in the second stomach, which is generally absent in cases of ulcer in the abomasum.

**Treatment.**—The treatment for ulcers in stomach or

bowels must be first of all purely dietetic. Its chief object should be to guard the ulcer against irritation by coarse or indigestible food. Zippelius, therefore, recommends for cattle only liquid and well-cooked nourishment, and entire avoidance of substances which readily ferment, such as brewers' grains. To horses give meal and bran-mash; to dogs milk and raw eggs, with finely-chopped meat. Of medical remedies artificial Carlsbad salt is by far the best. It not only neutralises the excessive acidity of the stomach, but ensures disinfection and rapid emptying of the stomach. Use either the official preparation or prescribe it in suitable mixture, say—sulphate of sodium,  $2\frac{1}{2}$  drs.; chloride of sodium, 5 drs.; bicarbonate of soda, 75 grs.; giving to a horse  $1\frac{1}{2}$  oz., to cattle 3 oz., and dogs  $\frac{1}{2}$ —1 dr. per dose. Spirits of salt are contra-indicated. Zippelius remarks on this point that intestinal ulceration in cattle is probably that serious form of indigestion in which spirits of salt (hydrochloric acid) act as a poison! On the other hand, in obstinate cases one may make a trial of subnitrate of bismuth (2 to 8 grs. for a dog), or of nitrate of silver. In haemorrhage of the bowels prescribe styptic remedies (tannin and preparations of iron or lead). To check persistent vomiting in dogs, use the narcotics already advised for catarrh of the stomach, viz.: opium, cocaine and bromide of potash, also creosote, creolin, tincture of iodine, fragments of ice, etc.

#### HÆMORRHAGE OF THE STOMACH AND INTESTINES.

**Etiology.**—This may arise from the most diverse causes, which are not always easy to identify. In spite of the fact that such haemorrhage is only a symptom of some other disease, yet it is wise to treat it carefully and independently. It is understood, of course, that we speak only of cases in which a large discharge of blood is noted.

1. In many cases haemorrhage is caused by mechanical or chemical injury to the gastro-intestinal mucous membrane. Such injury may arise from nails, bone-splinters or glass, fish-bones, sharp pine-needles, etc.; also from entosoa (larvae of *Gastrophilus*, *Tænia echinococcus*, *Echinorrhynchus gigas*); from irritants, such as undiluted tartar emetic; and, lastly, from concussion of the stomach produced by a kick, a fall or over-exertion.

2. Ulceration of stomach or intestines, especially the peptic form, may produce bleeding by opening the blood vessels.

3. Hæmorrhage very often occurs in the course of acute inflammation of the mucous membrane, as, e.g., in the various forms of enteritis and in the distemper of dogs.

4. Venous congestion of the mucous membrane of the stomach or bowels is not seldom followed by hæmorrhage. Such congestion ensues upon displacement of the bowels, as well as disorders of the heart, lungs or liver (valvular defects, emphysema of the lungs, induration of the lungs, cirrhosis of the liver, fatty degeneration of the same, or thrombosis of the portal vein).

5. Diseases of hæmorrhagic nature are sometimes accompanied by intestinal hæmorrhage. Among these are certain forms of anthrax (hæmorrhagic intestinal anthrax), petechial fever in horses, septicaemia, and septicæmic puerperal fever in cattle.

6. Hæmorrhage of the rectum arises from injuries, tumours and haemorrhoids. The latter occur as diffuse or nodular varicose enlargements of the haemorrhoidal veins, partly in the submucous (so-called internal haemorrhoids), partly in the subcutaneous tissue outside the anus (so-called external haemorrhoids). They are most frequent in swine, rarer in dogs, horses and cattle. They arise after general congestion of the circulation (heart, lung or liver complaints) through insufficient emptying of the haemorrhoidal veins, which are furthest from the heart of all the veins in the abdominal cavity. In pigs the evil is possibly also hereditary. Over-feeding with rich, indigestible or irritating food is also sometimes blamed, or lack of exercise, excessive use of drastic purgatives, etc. Enzootic inflammation is occasionally found as the cause of hæmorrhage of the rectum in cattle. The so-called "back or loin blood" was earlier attributed by Rychner (Bujatrik) to haemorrhoids, with which Spinola declared it to be related; whereas Haubner-Siedam-grotzky considered that it was caused by sharp, irritating and too highly seasoned food, or by over-exertion, which produced inflammation of the mucous membrane of the rectum. According to this, "loin-blood" is no other than a hæmorrhagic proctitis. In other cases, anthrax is the cause.

7. Vogel mentions a peculiar kind of hæmorrhage of the stomach. An aneurism of the abdominal aorta in a dog had grown fast to the stomach, and, on rupturing, emptied itself into the stomach, causing speedy death. A similar case was noticed in a mare by Labat and Cadéac, in which an aneurism of the aorta had emptied itself into the colon. During its last

few days the mare passed bloody faeces and showed signs of colic.

**Symptoms.**—The most prominent sign of stomachic haemorrhage is the vomiting of blood (haematemesis). A large amount of lumpy, dark-coloured blood mingled with food is discharged during repeated attacks. This symptom may, however, be absent, in which case the haemorrhage of the stomach is hardly distinguishable from that of the bowels. The latter is recognisable by the passage of faeces, which are either mixed with blood, or discoloured and tarry. At the same time, the signs of internal bleeding or chronic anaemia are present, and may lead later to hydrexia and serous transudation into the cavities of the body.

The phenomena of haemorrhagic inflammation of the rectum in cattle are sudden feverish attacks, stiffness of attitude, sensitiveness about the croup, straining to evacuate, which, however, only results in the passage of a few dry, blood-mingled faeces; also enhanced temperature, puffy swelling of the mucous membrane of the rectum, so that the hand, on being inserted, is made bloody. Recovery usually follows after three to five days, but where the inflammation is intense, the end may be fatal.

Was noticed in a horse, along with two clumps of piles the size of a walnut, frequently recurrent colic, with discharge from the anus of dark, fluid blood and occasional dark blood clots, followed later by anaemia and hydrexia combined with bleeding of the skin (so-called bloody sweat), which eventually caused death.

**Differential Diagnosis.**—It is not difficult to differentiate haemorrhages due to gastro-enteritis from those due to proctitis or haemorrhoids, but it is frequently difficult and even impossible to diagnose or locate with certainty such conditions as ulceration, irritation by foreign bodies, chronic congestion or hyperæmia. It is often important, in cases of discharge of blood from the mouth, to decide whether one has to deal with haemorrhage of the lungs or of the stomach. The latter is indicated by previous and subsequent gastric disorders, by distinct retchings, disgorgement of the contents of the stomach, the appearance of tar-like faeces, absence of cough and soundness of the lungs. The condition of the vomited blood is also an indication, as in haematemesis it is mostly clotted, dark in colour and showing acid reaction owing to the presence of gastric

juice. In contrast to this, the blood discharged in haemorrhage of the lungs is bright red has an alkaline reaction, and, owing to admixture of air, is frothy. Moreover, both before and after the haemorrhage, difficulty of breathing and coughing will be observed. Nevertheless, the differentiation may often be very difficult, when, for example, the blood coughed up is swallowed and afterwards vomited, or when, in haematemesis violent coughing accompanies the retchings. Spinola mentions a case in which vomiting in a dog after eating a quantity of blood was mistaken for haematemesis. Finally, haemorrhoids in dogs are often confounded with over-filling of the anal pouch and formation of abscesses therein.

**Treatment.**—This consists in the internal administration of haemostatics. To these belong cold water, small pieces of ice and astringents, viz.: alum, tannin, sulphate or perchloride of iron, sugar of lead, nitrate of silver, opium and ergot of rye.

## GASTRO-ENTERITIS.

**Classification.**—Inflammation of the gastric and intestinal mucous membranes of the domestic animals may be variously classified. In this chapter we shall consider, first the primary and independent inflammations, not those which are secondary, and which arise in the course of other diseases, such as rinderpest, influenza, dysentery, swine-fever, rabies, petechial fever or displacement of the bowels. An attempt has been made to divide gastro-enteritis, according to the chief seat, into an inflammation of the stomach "gastritis," and of the bowels "enteritis," and to subdivide the latter on the same principle into duodenitis, ileitis, typhilitis, colitis and proctitis. But in practice this can seldom be done, as both stomach and bowels are usually simultaneously affected. Rather might one take the degree of inflammation as a basis for classification and speak of parenchymatous, phlegmonous, suppurative, haemorrhagic, croupy, diphtheritic or caseous inflammations. The lowest degree would be represented by gastro-intestinal catarrh. Even better would be an etiological classification into: 1—Inflammation caused by simple mechano-thermal irritants; 2—that produced by poisons (toxic); 3—Infectious inflammation (dysentery); 4—embolic; 5—tubercular; 6—mycotic (produced by moulds), etc., etc. To speak of diffuse or circumscribed gastro-enteritis, of sporadic or enzoötic, of acute or

chronic, is much too general a differentiation. For clinical purposes we advise a classification based partly upon the degree and partly upon the cause. We, therefore, distinguish the following as forms of gastro-enteritis which can be more or less well characterised as independent and individual diseases :—

1. Simple, Non-Toxic Gastro-Enteritis of various domestic animals.
2. Croupy Gastro-Enteritis, especially of cattle.
3. Mycotic Gastro-Enteritis (so-called Typhus).
4. Toxic Gastro-Enteritis.

#### I.—SIMPLE, NON-TOXIC GASTRO-ENTERITIS.

**Etiology.**—The causes of simple ordinary gastro-enteritis differ from those which produce acute gastro-intestinal catarrh only in degree. Among these are violent internal or external chill, scalding of the gastric mucous membrane by hot drink (*i.e.*, by giving calves their milk too hot), irritant food, foreign bodies, and intestinal worms, especially the spiroptera species (sp. *megastoma* in horses; sp. *sanguinolenta* in dogs; sp. *strongylina* in pigs, etc.). Hay-mites (*Acarus foenarius*) have been charged with causing gastro-intestinal inflammation in horses, but probably unjustly, as they are more or less present in all hay. With regard to gastro-enteritis due to coccidiae and gregarines, compare the chapters on coccidian dysentery (bloody flux), coccidiosis of the intestines and liver, and also on diphtheria in birds.

**Pathological Notes.**—The symptoms of gastro-enteritis differ from those of acute catarrh only in intensity. But occasionally one form passes into the other and they become difficult to distinguish. In inflammation, however, we find haemorrhage instead of hyperæmia, and parenchymatous, phlegmonous, serous, or suppurative infiltration of the mucous membrane, even necrosis, instead of superficial desquamative processes.

1. Examination of the stomach reveals considerable redness, either circumscribed or diffuse, and varying in shade from light to dark. The mucous membrane is dull and swollen, sometimes in folds and infiltrated with blood, or covered with petechial spots or larger dark-red patches. The surface of the membrane is sometimes smooth, at others covered with tough or thin, clear or blood-coloured slime, and again it reveals small, round or longish, superficial ulcers

in greater or less number, whose edges are sharp and even, their base at times smooth and at others covered with blood coagulae (haemorrhagic erosion). These occur most frequently upon the folds and in the pyloric region of the stomach, and are most frequent in dogs and in the abomasum of cattle, where they are induced by haemorrhagic infiltration of the mucous membrane, with consequent superficial necrosis. The surface of the membrane is moreover roughened by the swollen and fatty disintegrated peptic glands, which project in the form of granules. Sometimes the submucosa is infiltrated with oedematous or purulent fluid.

2. In the bowels the mucous membrane is also red, swollen and dull, usually to a limited extent, but sometimes all over, or in dots, tooth or star-shaped or like net-work, and at times is marked with light or dark-red spots, stripes or patches. It is also infiltrated with fluid, soft, tender and disintegrated to a pulp, or like tinder, occasionally covered with dry, discoloured gangrenous scabs, which are either firmly fixed, or have been partly loosened by suppuration, and may have caused complete perforation of the intestinal wall. The epithelium of the mucous membrane is frequently dislodged over a considerable area, and the tufts of villi are swollen and prominent, appear larger, float plainly under water and give to the mucous membrane a velvety appearance. The solitary and agminated follicles are sometimes found unchanged, and at others swollen and surrounded by a patch of red, the solitary ones appear like nodules the size of a pin-head, the agminated ones larger and irregular. On disintegration of these, crater-shaped follicular ulcers remain. The contents of the bowels vary much in nature, being sometimes thin and watery, sometimes slimy and putrescent, at others clear, or milky, greyish or stained with blood, or mixed with epithelial flakes, coagulae of fibrin, small lumps of pus or blood, or they may even consist of blood alone. The submucosa is either normal or highly injected and oedematous, gelatinous or haemorrhagic and swollen, making the mucous membrane appear extremely thick and puffy. Thus, in the blind-gut and colon of the horse it may be found several fingers thick over a considerable surface. The muscularis is usually only slightly affected by serous infiltration, unless it be perforated or deeply ulcerated. The serosa is injected, and often shows a coating, as well as peritonitic proliferations. Finally, the mesenteric glands are frequently infiltrated and swollen, and the liver and spleen very full of blood.

**Symptoms.**—The phenomena of simple sporadic gastro-enteritis either follow gastro-intestinal catarrh, or appear suddenly and independently, which is most frequently the case. Appetite and rumination are at first entirely suppressed. Swine and dogs often vomit frequently, and both horses and cattle are nauseated. The membrane of the mouth is very red, hot and dry, and in later stages intense thirst is developed, the satisfaction of which may cause increased vomiting, especially in dogs. To this are added continuous and generally very violent symptoms of colic, which in dogs may amount to frenzy. The stomach and abdomen are painfully sensitive to touch, the abdomen is tense and sometimes tympanitic. Peristalsis is suppressed, and obstinate constipation ensues, during which the animals pass at most a few hard, small balls of faeces (after much straining), which are covered with mucus or blood, or they pass pure blood (haemorrhagic enteritis). In exceptional cases gangrenous foetid sloughs of intestine are passed. It is not until near the fatal termination that the constipation changes to severe diarrhoea, when to the paralysis of the intestinal walls is added that of the sphincter ani. The urine frequently contains albumen and, in herbivora, acid phosphates. The severe gastric attacks are accompanied by fever no less severe. This is indicated by a very quick, small, hard pulse, great rise of body temperature, greatly reddened mucous membranes, coldness of the extremities and sweating. In horses the pulse may be 80 to 100 beats a minute, and the internal temperature  $104^{\circ}$  to  $106^{\circ}$  F.; with the approach of death the temperature becomes sub-normal. The general sensibility is often very greatly diminished; it shows great fear and restlessness, or the excitement yields to a gradually increasing stupor and collapse, it sways and staggers and has occasionally convulsions and spasms. Towards the end the colic pains usually abate, and the animal expires with the appearances of coma.

The progress of simple gastro-enteritis is very rapid in the case of the horse, so that its duration seldom exceeds two or three days. The chronic form, characterised by recurrent attacks, usually lasts several weeks and terminates in death. It presents the same symptoms in the horse as thrombo-embolic enteritis. Gastro-enteritis occasionally occurs en-zootically.

**Prognosis.**—The disease is very serious and generally ends in death. Favourable appearances are—moderate symptoms of

colic, low fever, especially a strong and steady pulse and regular evacuation. Unfavourable, on the contrary, are—very high fever, a much accelerated, small and wiry pulse, dyspnoea, obstinate constipation, sudden cessation of pain, accompanied by collapse and profuse diarrhoea. Death follows probably from necrosis of the bowels with consequent general sepsis, or from apoplexy of the brain, oedema of the lungs, intestinal haemorrhage, or paralysis of the heart caused by shock.

**Differential Diagnosis.**—1. To distinguish gastro-enteritis from gastro-intestinal catarrh, one has to be guided chiefly by the intensity of the inflammatory symptoms : high fever, weak action of the heart, general condition, severe colics, and constipation, followed by passage of blood.

2. In horses it is more difficult to distinguish from other forms of colic a *gastro-enteritis* which first develops as a so-called "Inflammatory Colic." In fact, it is often impossible during life. A further great difficulty lies in the fact that many a colic terminates in enteritis. And yet a differential diagnosis should in every case be at least attempted. Guiding indications are : high fever, rapid, small pulse, intermittent colicky pains, general debility, prolonged constipation, painful sensitiveness of the abdomen to pressure and the previous history of the case. Occasionally a diagnosis may be made by exclusion.

3. From the remaining forms of *gastro-enteritis* the foregoing may, in general, be distinguished by observing the following signs :—In the toxic form the previous history of the patient and the characteristic signs of certain poisons ; in mycotic cases the peculiar nervous phenomena ; in the croupy form the passage of fibrinous membranes and its longer duration ; in infectious dysentery its enzoötic spreading and the prevalence of diarrhoea.

4. A primary *gastro-enteritis* may be differentiated from rabies, anthrax, rinderpest, etc., by careful examination and watching the progress of the disease.

**Therapeutics.**—After the removal of the exciting causes, the treatment of simple *gastro-enteritis* demands careful watching and regulation of diet. At first complete abstinence is to be advised, followed by light, digestible food. Harm is occasionally done by giving too soon food difficult of digestion. The water must be tepid. To dogs give as little water as possible, as drinking produces vomiting.

The further treatment begins with restoration of activity to the skin by friction, rubbing the abdomen with spirits of camphor, turpentine, or mustard oil, and applying wet cloths. Inwardly administer soothing remedies, especially mucilaginous decoctions and oil-emulsions, such as linseed, fresh linseed-oil, barley-water and mucilage mixed with opium, tincture of opium, etc. If the colicky pains are violent, use injections of morphia. Caution is necessary in using drastic remedies against obstinate colic, owing to the risk of increased irritation of the mucous membranes. Use, therefore, only the mildest purgatives, such as castor oil in emulsions, Glauber's salt and sulphate of magnesia, as well as calomel, the latter often in combination with opium.

As examples, the following recipes may be useful :—

For the horse : An electuary composed of powdered opium,  $2\frac{1}{2}$  drams; calomel, 30 grains; powdered marshmallow, 3 ounces; distilled water q.s. (one dose).

For the ox : Decoction of linseed (3 ounces in a pint of water) : linseed oil, 10 ounces.

For the dog : Tincture of opium, 30 to 75 minims; mucilage of gum arabic, 5 ounces; water, 5 ounces (dessert-spoonful doses).

In great weakness or collapse prescribe camphor, ether, wine, coffee, caffeine, hyoscyamus, etc.

## 2.—CROUPOUS ENTERITIS.

**Occurrence.**—Croupous enteritis is most frequent in cattle, in which it is far more common than is generally supposed. It is uncommon in the horse, more so in the dog, very rare in the sheep. It is sporadic in the ox and has always a chronic course. In the other animals it is also generally sporadic, but it may occur in either acute or sub-acute forms.

**Etiology.**—Cold is generally believed to exert some influence on its production, particularly as it is frequent in the spring and autumn. Irritating and indigestible foods, too, undoubtedly are frequently responsible; hay with a large admixture of aromatic herbs; overfeeding with chaff. In some cases it has been traced to injudicious use of drastic purgatives. Other drugs, such as spirits of camphor, cantharides, etc., may also produce croupous enteritis in cattle. The reason why this complaint is chiefly noticed among cattle is that they are sup-

posed to be naturally predisposed to it, which fact may rest on their blood containing much fibrin and albumen, with tendency to fibrinous excretions. This is especially marked in young and well-nourished animals, and in those advanced in pregnancy, which suffer from intestinal croup with great frequency as compared with others. Perhaps, also, the lymphatic constitution peculiar to cattle may have some connection with it, one consequence of which, for example, is that in inflammation of the lungs enormous plastic exudations of that organ so readily occur.

In horses coprostasis appears sometimes to cause intestinal croup, and tape-worms in dogs and cats, but frequently the cause is not determined.

**Pathological Notes.**—The seat of croupy inflammation in cattle is either the small intestine or the colon ; in horses, usually the small intestine. The mucous membrane presents catarrhal or inflammatory changes in different degrees, and is covered to a greater or less extent with greyish-yellow croupy matter, more or less easily detachable, beneath which the membranous tissue is very red, soft and friable and infiltrated with blood and pus. This croupy matter sometimes forms itself in cattle into stratified layers, and at others it is tubular, cord-like, or cylindrical, and either solid or hollow and filled with food. Occasionally it consists of several tubes, one within the other, which can be drawn out, and between which particles of food may be found. The length of these tubes varies very considerably ; at times they are only short, but occasionally measure as much as ten yards. Microscopic examination reveals an amorphous structure of fibrinous matter, with embedded, fatty-degenerated epithelial cells, and white blood-corpuscles. The contents of the intestine consist either solely of this croupy matter, or in addition a thin, fluid substance, often smelling very badly and discoloured with clotted coagulae. On closer examination the tubular glands of the intestine are also found to be filled with fibrinous exuded matter.

**Symptoms.**—The symptoms of croupous enteritis in cattle are much milder than those of other forms of enteritis. Indeed, occasionally no marked phenomena whatsoever are noticeable ; at other times they only appear in connection with the expulsion of croupy matter. Usually the signs which precede the evacuation of the above-named fibrinous formation are merely

those of intestinal catarrh or of chronic enteritis. Disturbance of appetite and of rumination are noticed, with slight colic, constipation and low fever. After a few days the croupy discharge begins, combined generally with severe diarrhoea and passage of thin, foetid, greyish-brown faeces, mixed with fibrinous shreds and occasionally infiltrated with clots of blood or pus. This evacuation is usually followed by improvement, often by extremely rapid recovery, so that the entire duration may in general be six to eight days. In exceptional cases the disease may be more severe in cattle. Thus Festal records an acute croupy enteritis in the ox, which commenced with frequent colic, obstinate constipation, high inflammatory fever and tottering gait, in which, after abatement of the colicky symptoms, but with continuous constipation, membranous masses were squeezed out, followed six or seven days later by an ichorous, evil-smelling fluid. Then came improvement, or else death ensued after general increase of debility and formation of peritonitis. (Duration, eight to twenty days.)

Croupy enteritis generally manifests itself in horses by symptoms of persistent indigestion-colic. Prietsch reports in one case maniacal signs (kicking, biting, etc.). Sometimes these colicky symptoms are lacking, and the ailment proceeds essentially as a feverish affection of the bowels, with discharge of soft, foetid faeces, mingled with flaky, clotted or stringy, croupy matter. In other animals the appearances resemble those of simple gastro-enteritis, but are less severe.

**Prognosis.**—This is rather favourable for cattle. In rare cases, danger may arise from complete stoppage of a bowel-section by croupy matter. In other animals, especially horses, the course is likely to be more serious.

**Differential Diagnosis.**—The passage of ropy or tubular croup-substance through the anus, which alone renders diagnosis of bowel-croup possible, may lead to error, especially with the untrained. Thus these bands have often been mistaken for snakes or tape-worms, for intestines of smaller animals, which have been swallowed, or for intestinal loops of the patient itself. But a careful examination of these croupy cylinders, their greyish-white colour, homogeneous structure, the absence of mesenteric adhesions, or of any definite arrangement of blood-vessels, will easily prevent their being mistaken for intestines. In horses, slime-covered dung-balls, such as are often seen in

ordinary constipation or catarrhal proctitis, must not be confounded with the products of bowel-croup. And in carnivora also the same mistake must not be made in the faeces containing mucus or half-digested connective tissue-like membrane, which proceeds from the food.

**Therapeutics.**—The treatment of croupy enteritis is not essentially different from that of the simple form. Only solvent and liquefying remedies are to be preferred to emollient ones. The neutral salts, and particularly the alkalis, whose object is to dissolve the croupy coagulae (bicarbonate and sulphate alkalis) are useful. These salts may also be administered in diluted form in enemas, particularly for dogs (a 1 per cent. solution of common salt, soda or potash).

#### MYCOTIC GASTRO-ENTERITIS.

(*Infectious, septic, septiform, typhoid gastro-enteritis; intestinal mycosis; mycosis or sepsis intestinalis; enteric fever; and poisoning by meat, fungi, or sausages.*)

**Definition.**—All the above-named conditions are included under the name of mycotic gastro-enteritis. From the etiological point of view they are similar, all being caused by the irritation of some injurious material, affecting first the intestinal canal and secondly the whole system. The active principles must be regarded as operating in a two-fold manner: first, *septic infection*, that is an invasion of micro-organisms; and secondly, as a *septic intoxication or poisoning*, due to the absorption of the chemical products of those micro-organisms. These chemical substances, the result of bacterial metabolism, are called "Ptomaines," "Toxines," "Corpse-Alkaloids," etc.

Mycotic gastro-enteritis may therefore be more exactly defined as an intestinal affection, accompanied by general indisposition, and resulting from consumption of decayed foods, partly through the invasion of pathogenic microbes and partly through absorption of poisonous ptomaines. It lies midway between an infectious disease and toxic gastro-enteritis, possessing features common to both. There is a distinct connection between meat-poisoning and septicæmia on the one hand, and the poisonings due to fungi and acrid narcotics on the other.

The fact that mycotic gastro-enteritis is distinguished not only by genuine gastric disturbances, but also by grave

general symptoms, such as great debility, cerebral disturbance and a rapid course, led in earlier times to its being called "typhoid" gastro-enteritis, or "bowel-typhus." It was erroneously believed to be identical, or at least analogous to, enteric fever in man.

In the following we describe, first, such mycotic gastro-enteritis as is caused in carnivora, omnivora and birds by the consumption of decomposed flesh; and then the similar ailments noticed in the herbivora after eating fungous vegetable matter (mould-fungi, uridines, or rust and smut-fungi). With regard to the latter we must observe that the kind of fungus cannot always be exactly determined, and that there are outbreaks which offer the typical aspects of fungous poisoning, although one has not been able to prove that the fungi are the direct cause of illness. Sometimes, too, in cases of fungus poisoning, all signs of inflammation in the gastro-intestinal mucous membrane are absent, because the poison passes directly into the blood, without injury to that membrane, producing essentially nervous symptoms.

**General Notes on Ptomaine.**—By ptomaines (ptomatinæ toxines, corpeo-alkaloids, septicines) we understand generally the metabolic products of bacteria both within and without the body. These arise, not only in dead, but also in living bodies. The latter have been distinguished by the name of leucomaines (leucomatine). In their chemical nature toxines are not bases alone, as was long supposed (corpeo-alkaloids), but also albuminous substances (toxalbumine), acids, etc. The very varied character of their chemical composition is shown by Kobert in great detail in his "Text-Book on Poisoning" (1893).

#### I.—MYCOTIC GASTRO-ENTERITIS OF CARNIVORA, OMNIVORA, AND BIRDS AFTER EATING DECOMPOSED FLESH. MEAT-POISONING.

**Etiology.**—Mycotic gastro-enteritis in our flesh-eating domestic animals is closely allied, both in origin and symptoms, to "meat-poisoning," as observed in man. Probably a great many of the cases formerly reported as "dog typhus" or "swine typhus" were mycotic gastro-enteritis. It is due to the consumption of meat in a more or less advanced state of decomposition, stale sausages, old herring-brine, or rotten cheese. It is most frequently seen in butchers' and shepherds' dogs. It has, however, been noticed in fowls after eating bad meat. The actual cause of the illness is to be sought in the bacteria present in the meat as the result of putrefaction, and in the ptomaines

to which they give rise. As usually little is known of the patient's recent history, the trouble is frequently mistaken for a case of mineral poisoning.

**Symptoms.**—1. *In meat-poisoning* these consist in a sudden, very violent and often bloody diarrhoea, vomiting, intense thirst, high fever ( $104^{\circ}$ - $107^{\circ}$  F.), extreme weakness and lassitude, followed quickly by collapse. Death often occurs in a few hours, usually within 24. Schindelka noticed haemorrhage of the retina in two dogs, by ophthalmoscopic means.

2.—*In sausage-poisoning* the same symptoms are present, but characteristic indications of paralysis are also often seen, such, for instance, as difficulty of swallowing, dilation of the pupils, ptosis (*i.e.*, paralysis of the upper eyelid), severe tympanites (paralysis of the stomach) and constipation (*ditto* of the bowels).

3.—*Fish-poisoning*, which occurs in swine after being fed with herring-brine, is characterised by spasms. The animals show cramp of the jaw, epileptic and tetanic convulsions, the phenomena of brain disorder, giddiness, and difficulty in swallowing, etc.

That the susceptibility of animals to this form of poisoning varies greatly has been experimentally proved by several enquirers. Thus Semmer found that dogs and cats, on being fed with the flesh of a horse that had died of septicæmia, remained in good health, whereas three pigs sickened and died. Colin, in similar trials, only noticed a very slight diarrhoea; Lemke, on the other hand, produced very violent diarrhoea in three young dogs by feeding them with flesh of animals that had died of anthrax, whilst an old dog displayed no morbid symptoms. Similar observations have been made with fungoid poisons in herbivorous animals.

**Post-mortem Notes.**—On examining the contents of stomach and bowels one will find half-digested, foul-smelling masses of flesh, chocolate coloured, bloody and of a mucous, fluid consistency; while the mucous membrane is swollen, considerably injected and haemorrhagically infiltrated, the follicles, both solitary and agminated, as well as the mesenteric glands are all swollen. The blood is altered; the liver enlarged and rapidly decomposing; the spleen swollen and infiltrated with haemorrhagic foci, while the muscular tissue of the heart is very friable.

**Meat and Sausage Poisoning in Man.**—Whereas at first these conditions of disease were attributed to infection by the flesh of animals suffering from anthrax, and the name of "Intestinal Mycosis" was supposed to be equivalent to intestinal anthrax, Bollinger has proved that the so-called mycosis of the bowels is not anthrax, but a septic inflammation of the intestinal mucous membrane caused by mycotic poisons which have not yet been isolated. But we must distinguish between ectogenous injuries of the flesh—i.e., such as only arise after death—and the endogenous defects, or those which occur in the living animal.

1. To the ectogenous causes of disease belong especially sausage poison and fish poison, with a few of the meat poisons. These ectogenous poisons are essentially bacteria, which are destroyed by boiling. The course of the disease is, consequently, a mycotic infection. These bacteria, however, generate chemical products of metabolism (ptomaine, toxine), which are not destroyed by boiling. Gärtnér observed, during a case of wholesale poisoning in Frankenhäusen, characteristic bacteria both in the poisonous meat and in the organs of a man who died from eating it, to which he gave the name of "*bacillus enteritidis*." These bacteria showed lively rotary movement in the suspended drop, took aniline colour very strongly at one end, while the rest seemed hardly coloured at all. Moreover, the meat contained poisonous matter which resisted boiling, and produced in animals inoculated therewith enteritis, convulsions and the symptoms of paralysis. Dogs and cats were immune to the bacteria, while rabbits, guinea-pigs and mice died with all the appearance of violent enteritis. Johne also met with the *bacillus enteritidis* in a case of meat poisoning at Bischofswerda. Gaffky and Daask discovered in a wholesale poisoning case at Röhrsdorf, caused by eating horseflesh sausages, characteristic "sausage bacilli," in the form of movable rod-shaped bacilli, with rounded ends, which sometimes developed into threads, and flourished best in slightly alkaline broth. Given to monkeys, guinea-pigs and mice, these sausage bacilli produced enteritis and diarrhoea. Dogs, cats, pigs and rabbits, on the other hand, seemed to be immune. Haupt says that he proved a case of meat-poisoning in Chemnitz to have been caused by a bacterium (*Proteus mirabilis*). Nauwerk also found in poisonous sausages a bacillus with wonderfully septic properties, which he believed had got into the sausage from the intestinal canal of the pig in question, in which also it was found. A similar bacillus was found by Moulé, which had a strong smell of rancid butter. Poels and Dhout found short and very fine rods in the marrow of a calf, which on inoculation produced enteritis and haemorrhage in both lungs, liver and spleen, where they generated a toxine which resisted a temperature of 280° F. From these and other reasons it is certain that the bacteria which cause poisoning vary very greatly, and that their products, the ptomaines, are also by no means chemically uniform, but differ according to the origin and nature of the flesh, and according to locality, conditions of temperature, degree of putrefaction, method of preparing the meat, the means adopted for its preservation, etc., etc.

The signs of sausage-poisoning (*botulismus, allantiasis*) are quite peculiar. Besides appearances of septic gastro-enteritis (vomiting, accompanied by diarrhoea, faintness, affection of sensorium and collapse) there are very special symptoms of paralysis; that is, paralysis of certain groups of

muscles : thus, for instance, of the eye (ptosis, or drooping of the upper eyelid) ; of the larynx (difficulty of speech in men) ; of the pupils (mydriasis) ; of the pharynx (difficulty of swallowing) ; of the intestines (constipation), etc., etc. Such partial paryses occur, as is well known, in the course of infectious diseases, such as diphtheria, rabies in dogs and milk fever in cattle. This must be regarded as due to the ptomaine.

2. The second group of meat-poisoning cases (endogenous) contains those caused by the flesh of diseased animals. Bollinger styles the illness thus produced in man, " sepsis intestinalis." To this belong the bulk of so-called meat-poisoning cases. As the poisonous agent consists partly of bacteria and partly of ptomaines, the poison is only weakened by cooking the meat, and not entirely removed. Such poisonings are most frequently caused by the flesh of septically or pyematically affected cows (*Septicæmia puerperalis*) and calves (*Polyarthritis pyæmica*) ; also by that of animals with ulcerous nephritis, septic affections of the liver or lungs, enteritis, peritonitis and mastitis. Strange to say, illness sometimes results from eating meat apparently quite normal both in appearance and taste. This is to be explained either on the ground that the disease was already incipient, or that the meat had been infected after slaughtering.

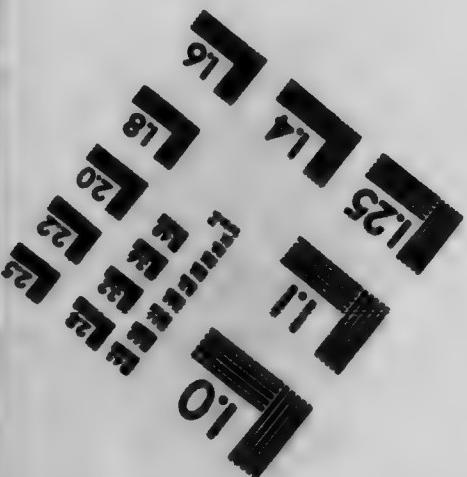
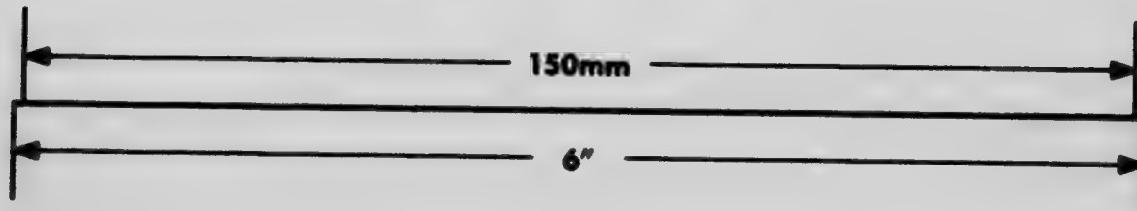
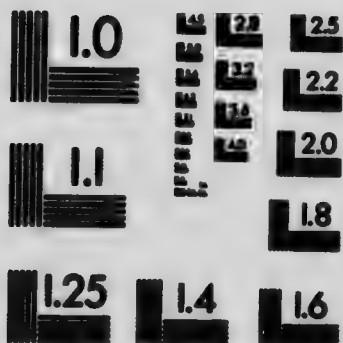
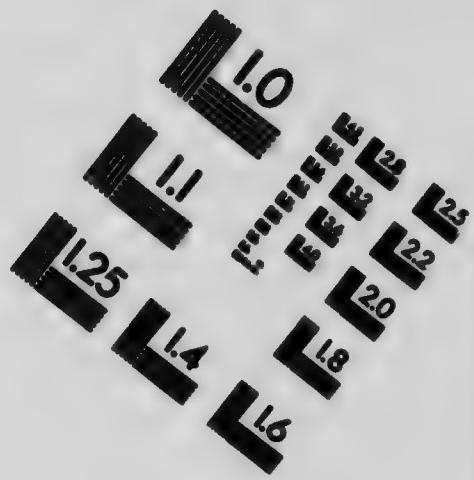
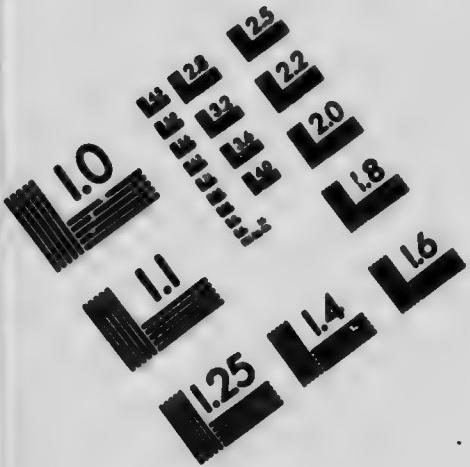
The phenomena of this meat-poisoning are those of septic gastro-enteritis. The symptoms are the same as with our domestic animals, viz. : vomiting, diarrhoea, colic-pains, high fever, great prostration, affection of the sensorium and collapse. In other cases there is only indigestion, or perhaps a gastric catarrh, or so-called gastric fever, or vomiting, with diarrhoea. Very often its course presents the appearance of cholera on the one hand, or of enteric fever on the other, or both combined. This fact has given rise to the supposition that perhaps abdominal or enteric fever may be caused by meat-poisoning. This is, however, not to be accepted, for the phenomena of enteric fever (and of cholera) can be produced by other morbid causes than the specific typhus bacillus ; and also because all attempts to transmit human enteric fever to animals by inoculation have failed, whereas they may be easily infected with meat-poisoning. In fact, during the different epidemics of meat-poisoning among human beings, similar disease has often been noticed in dogs, cats, pigs, fowls, etc.

## 2.—MYCOTIC GASTRO-ENTERITIS OF HERBIVORA, AFTER CONSUMPTION OF FOOD CONTAINING FUNGI (MOULD FUNGI, URIDINEÆ, USTILAGINEÆ). SO-CALLED POISONING BY FUNGI.

**Etiology.**—Under the names of enteric fever, typhus, gastro-enteritis, etc., we find among herbivorous animals a number of cases of disease described, which, on closer inspection, appear to be closely allied to meat-poisoning in dogs. To these belong first of all those remarkable and, at first, almost inexplicable ailments which were noticed in horses, cattle, sheep and pigs after eating damaged or rotten fodder (straw, hay, oats, oil-cake, chaff, turnips, potatoes, meal, bread). For instance, the so-called "shavings sickness" of cattle, which in



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North Germany followed in many districts from feeding them with putrid beetroot shavings from the sugar factories. Further, in cows and horses illness was caused by feeding on unsound, spoilt, and fermenting potatoes. There is no doubt that these are cases of the operation of infectious mycotic materials upon the intestinal mucous membrane, and of absorption into the blood of ptomaine-like bodies. Moreover, in our literature numerous instances are given of the injurious effect upon our domestic animals of the fungi which vegetate upon their herbivorous food, and the above-named cases of injury from bad, rotten or fermenting food can be attributed to the presence therein of fungi. The following species of fungus growth are to be considered :—

1. *Mould-fungi (mucorinae)*, and especially mucor, aspergillus, and penicillium, which, though in themselves not injurious, yet by inducing decomposition of food materials (bread, flour, oats, hay, straw) give rise to ptomaines.

2. *Uridinea*, of which puccinia graminis is the most important. The experiments made by Franck with uridinea on rabbits produced precisely the symptoms of meat-poisoning, viz.: gastric disturbances, with staggering and spasms.

3. *Ustilaginea*. The chief representatives of this group are the greasy smut of wheat (*tilletia caries*), which is clinically by far the most important, and also ustilago carbo (dusty or flying smut) and ustilago maydis (the smut of maize). The smut fungi (*ustilaginea*) have the most intense effect of all fungi upon the digestive mucous membrane and general organism. This powerful effect must be attributed to some irritant ptomaines which develops in the plant as waste products of the fungi.

4. *Germ-fungi (pyrenomycetes)*. Clinically important is *Polydesmus exitiosus*, the "rape destroyer," which feeds on the green parts of rape.

5. *The yeast-fungus*, the cause of alcoholic fermentation. This is probably the last stage of development of the mould-fungus.

**Symptoms.**—The aspect of poisoning by fungi is very varied, and its appearances in general as follows :—Illness appears suddenly and generally in several animals at once, which gives it the appearance of an epidemic. The animals forsake their food and drink, become restless, get abdominal pains, which may be shown by moans and even bellowing.

Peristaltic action is suppressed, and there is at first obstinate constipation, followed later by profuse diarrhoea, with discharge of stinking, bloody and slimy matter. Slight tympany is sometimes present. Occasionally the muscles of the tongue and pharynx are paralysed; hence comes a free discharge of saliva, continuous chewing and cough. These gastric phenomena are accompanied by high fever, with an abnormally quickened pulse (in cattle 100 beats and above), ague fits, palpitation, coldness of the extremities, etc. At other times there is extreme weakness and prostration, with general muscular paralysis (as though from hemlock poisoning), which is the most important symptom to note. The animals lie motionless on the ground in a paralysed condition. Sometimes cerebral symptoms are noted, such as dizziness, insensibility, behaviour suggestive of chronic shivering, amaurosis and cerebral spasms. Very frequently the chief symptom is nephritis and cystitis (polyuria), and sometimes abortion also. Many cases of fungus-poisoning are at last marked by tympanites, palpitation, dyspnoea, and others by inflammatory swellings of the skin of the head (mucous membrane of lips, mouth and nose). The duration of the disease is often very short (about a day); but not rarely lasts eight, or even fourteen days. Death frequently ensues with the appearance of apoplexy.

The great variety of these phenomena may perhaps be best explained by the fact that the metabolic products of mould-fungi vary greatly, both chemically and physiologically, according to the changing outward conditions (temperature, place of growth, degree of development, etc.) of the fungus. It is not impossible that the simultaneous drinking of warm water (the water of brewers' grains) may have a peculiar influence upon the development of the fungi, and therefore upon the aspect of the disease. This is in accordance with the fact that mould-fungi do not, under all circumstances and in all animals, produce any pathogenic effect. Possibly some predisposing cause is present in the digestive organs. As, for instance, in thrush (*aphtha*) all the animals in a stable are not affected, so it would appear that some are immune against mould-fungi. On this account experiments in feeding with this fungus have often given only a negative result. But to deny the pathogenic nature of mould-fungi on these grounds would be as unjustifiable as it is illogical.

*The aspect of individual cases of mycotic poisoning varies much, and is often very characteristic. Sometimes if several sorts of fungi are asso-*

ciated, the symptoms are irregular. Those which are peculiar to the various forms of fungoid poisoning are as follows :—

1. *Mould-fungi*.—Loss of appetite, colic, tympanites, constipation, diarrhoea, blood-stained, slimy and sometimes very malodorous faeces, polyuria, dizziness, insensibility, conduct as in chronic hydrocephalus, paralysis of limbs, tongue, etc., and amaurosis. Also profuse sweating.

2. *Smut-fungi (Ustilagineæ)*.—Flow of saliva, persistent chewing, staggering, reeling and general paralysis of locomotion and sensation. In other cases, gastric symptoms.

3. *Rust or mildew-fungi (Uridineæ)*.—Inflammation of the skin of the head (lips, cheeks, eyelids), conjunctivitis, urticaria, stomatitis, pharyngitis, glossitis, colic, bloody diarrhoea, haematuria, paralysis and somnolence.

4. *Germ-fungi (Pyrenomyces)*.—Similar appearances; stomatitis, pharyngitis, rhinitis, dermatitis on the head, gastro-enteritis and paralysis.

5. *Yeast-fungi*.—Considerable cerebral excitement with subsequent stupor and signs of paralysis.

*For further details on the various fungid poisons, see Fröhner's "Text-Book of Toxicology for Veterinary Surgeons."*

**Post-mortem Notes.**—As with the external symptoms, so also there is considerable variation in the *post mortem* lesions. As a rule, one may expect the signs of severe haemorrhagic gastro-enteritis, with general septic changes (swelling of the liver and spleen, fatty degeneration of the muscles of the heart). But often the result is purely negative, as in narcotic poisoning. The following is to be noted concerning the various forms of fungoid poisoning. With *mould-fungus* one finds inflammation, ecchymosis and erosion of the gastro-intestinal mucous membrane, as well as hyperæmia and oedematous infiltration of the brain and spinal cord. In poisoning by *ustilagineæ* a *post mortem* examination yields generally negative or very slight results. Sometimes haemorrhagic gastro-enteritis is found, as well as streaks of sooty discolouration on the mucous membrane of the smaller intestine (so-called eel-skin). Dissection in cases of poisoning by *uridineæ* and *pyrenomyces* reveals dermatitis, stomatitis, pharyngitis, haemorrhagic gastro-enteritis, nephritis and cystitis. In consequence of the frequent association of several fungi, the *post mortem* results are far from being uniform.

**Differential Diagnosis.**—Mycotic inflammation of the bowels is frequently confounded with other complaints owing to its peculiar septic-infectious character. The following must be noted as likely to be mistaken for it :—

i. *Various toxic gastro-enteritis*, with similar rapid course

and the same grave general affection. Differentiation is only possible by discovery of the cause.

2. *Rinderpest*, especially when several animals are simultaneously struck with intestinal mycosis from eating the same damaged fodder. But certain symptoms peculiar to rinderpest will be absent, viz.: widespread inflammation of all the mucous membranes (not only those of the digestive organs, but also of the breathing apparatus, eyes and vagina), the characteristic erosions on the membrane of the mouth and vagina, its terrible contagiousness and the *post mortem* results.

3. *Anthrax*. This is recognised by its rapid course and by bacteriological demonstration of the specific anthrax bacillus.

4. *Foot-and-mouth disease*. Poisonings caused by *Uridinea* and *Pyrenomyces* (*Stomatitis ulcerosa*) are particularly apt to be mistaken for this disease. They were formerly designated sporadic thrush (*Aphtha*), but are easily distinguished from foot-and-mouth disease by their non-contagious character.

5. *Pox*. Poisonings by the two last-named fungi may in sheep be confused with pox, owing to the inflammatory skin affection of the head which they produce. But the lack of contagiousness, as well as the absence, for the time being, of sheep-pox in the country help one to a sure diagnosis.

6. *Dysentery*. This infectious form of gastro-enteritis is accompanied from the beginning by diarrhoea, generally lasts longer and has rather the character of contagious epidemic disease.

7. *Subacute inflammation of the brain* in horses and *cerebro-spinal meningitis* in cattle. In most cases bowel symptoms are absent. Yet in many instances of inflammation of the brain in horses dissection has revealed septic changes of the internal organs, as well as gastric disorder, suggesting resemblances to mycosis or infectious disease.

8. *Rabies*. The differential diagnosis—often not otherwise easy—depends, in the first place, upon the history, and next upon the prevalence of bowel symptoms at the commencement of the mycotic gastro-enteritis.

9. *Influenza*. This is distinguishable from the form of enteritis just named by its contagiousness, and its quite specific symptoms, particularly the affection of the eyes. But in some cases differentiation is not easy.

**Treatment.**—With regard to the treatment of mycotic enteritis in different animals, we recommend emetics for

dogs at the beginning, and later on purgatives, especially calomel. The latter is also good for horses, owing to its disinfectant qualities. For ruminants saline remedies are preferable. In dogs a washing out of the bowels with warm solutions (1 per 1,000) of tannic acid, or a weak solution of creolin, is useful. But very often the best result is obtained with stimulants (wine, spirits of wine, ether, veratrine, atropine, caffeine or camphor). General treatment is the same as that already given for gastro-enteritis.

**NOTE.**—The etiology of mycotic gastro-enteritis is often very obscure. Cases often occur among domestic animals with all the signs of fungoid poisoning, in which, nevertheless, the fungi cannot be found, or at least not in quantities sufficient to account for the disease. Much has yet to be made scientifically clear on this subject. We should like especially to point out that in poisoning by *Tilletia caries* and *Puccinia graminis*, the spores of these fungi are often found in fodder which is thoroughly wholesome. (See the tests made by Pusch.) It is not improbable that many of these cases presumed to be fungoid poisoning of herbivora really represent infectious diseases of which the nature and cause are yet unknown. (Compare the cases published in recent years by Schmidt, Gröninger, Louis, Munkenbeck, Albrecht, Siecheneder, Wilhelm, Bayne, Gotteswinter, and others.)

The gastro-enterites formerly styled "typhus" have nothing in common with human typhus. In part, they are to be regarded as fungoid poisonings. Thus Gerlach describes a "typhus" inflammation of the mucous membrane of stomach and bowels in horses, accompanied by colic, constipation, quick feeble pulse, grinding of teeth, etc., and essentially different both from ordinary colic and from the so-called gastric-bilious form of influenza and from anthrax. As causes, Gerlach cites feeding with fresh and partly unripe oats, not properly dried, and with badly-conditioned straw (infected with fungi) instead of hay. Derache also reports a similar non-contagious sickness among horses as "typhus," which yet had nothing in common either with petechial fever or with anthrax. Most of the outbreaks affecting many horses in the same stable offered no etiological points beyond bad and stinted feeding with dusty, damp, evil-smelling hay and oats of the poorest quality. The symptoms were those of mycotic gastro-enteritis, viz.: sudden attack, paralytic weakness, extremely feeble pulse, retarded evacuation with later diarrhoea, discharge of slimy feces and rapid fatal ending. On dissection quite characteristic changes were found, which reminded one very much of the abdominal typhus (enteric fever) in man, viz.: great swelling of the solitary follicles and Peyer's plaques, with putrid infiltration, ulceration and formation of scabs to the point of perforation, extensive ecchymosis, dendritic vascular injection as well as grey and brownish discolouration, pulpy, sticky and putrescent intestinal contents. The body rapidly decomposed; its flesh was soft and discoloured, the liver friable, the heart as though boiled, and the blood refused to coagulate and contained bacilli. Attempts at infection and even intra-venous inoculation proved ineffective. There was complete absence of swelling of the spleen, or car-

buncular swelling of the subcutis and intestinal submucose. This case presents much resemblance to human enteric fever, yet cannot be identified with it, as no conclusive proof is shown, and all attempts hitherto made to transmit the enteric fever of mankind to animals (cattle, calves, pigs) have failed. An undoubted clinical case of enteric fever in our domestic animals has not yet been observed.

### 3.—TOXIC GASTRO-ENTERITIS (POISONING).

**General Remarks.**—Toxic gastro-enteritis may be caused by numerous poisons, which may be divided into two great groups :

1. Acrid poisons.
2. Acrid narcotic poisons.

Toxicology being of great importance to the practitioner, we have preferred to deal with each of the acrid and narcotico-acrid poisons separately rather than enter upon a general disquisition on "toxic gastro-enteritis." This we do as briefly and in as practical a form as possible.

But first of all, a few remarks on the diagnosis of such cases are indispensable. It is not always easy to recognise a case of poisoning, as the symptoms in life and after death often bear great resemblance to those of infectious diseases, as well as to non-toxic gastro-enteritis. In general the following points are important aids to diagnosis :—

1. *The previous history of the case or patient.*
2. *Suddenness of attack, rapid course and often sudden death.*
3. *Connection between the symptoms and the food taken.*
4. *Simultaneous sickness of many animals without demonstrable contagion.*
5. *Complication of gastric and nervous symptoms.*
6. *The characteristic symptoms of certain poisonings and the pathological lesions.*
7. *Physical, chemical and pathological proof of the presence of the poison.*

### I.—MINERAL POISONS.

#### POISONING BY PHOSPHORUS.

**Symptoms.**—Attacks of colic, vomiting, groaning, icterus, albuminuria, sometimes inability to swallow, with swelling of the tongue; paralytic appearances, weakness of

heart, sub-normal body temperature; trembling; accelerated pulse and breathing; and in prolonged cases haemorrhagic diathesis. After inhaling phosphorus vapour: cough, attacks of choking, emphysema in the lungs, skin and on neck and thorax. In *Birds*: faintness, prostration, thirst, diarrhoea, and at times remarkable jumping movements. Duration: from a few hours to several days. If the sickness be prolonged (say three to five days), a remission of the severer signs often takes place, with apparent improvement. Schindel'ka noticed with a number of milch-cows that after complete recovery there was an *entire cessation of milk*, so that they had to be slaughtered.

**Post-mortem Lesions.**—Very similar to those of arsenical poisoning, viz: gastro-enteritis (which may, however, be absent), in certain cases even stomatitis and pharyngitis; glandular gastritis (parenchymatous degeneration of the glands of the stomach); if the affection last many days, fatty degeneration of the internal organs supervenes, particularly of the liver, heart, epithelium of the kidneys and muscles; bronchitis on breathing phosphorus vapour; ecchymosis and haemorrhagic foci in the various organs, dark, fluid blood, phosphorescence of contents of stomach and bowels with emission of vapour and a characteristic smell of phosphorus.

**Therapeutics.**—Administration of emetics, especially sulphate of copper (to form insoluble compound with the phosphorus), permanganate of potash, in 1-5,000 solution, peroxide of hydrogen in 1-3 per cent. solution, nitrate of cobalt, old spirits of turpentine rich in oxygen (given with large quantities of mucilage) to oxydise the phosphorus into non-poisonous phosphoric acid. As a dose for horses give 1½-3 ounces of the spirit of turpentine; for cattle 3-6 ounces; for pigs 6-12 drams; for dogs 1-2½ drams; and for fowls 5-10 drops; also stimulants. Oils must be carefully withheld as they would only dissolve the phosphorus and render it more active; the same remark applies also to milk.

#### ARSENICAL POISONING.

##### (A)—ACUTE FORM.

**Symptoms.** — These are primarily of gastro-enteritic nature; vomiting, salivation, colic, constipation; in

cattle, tympanites and later stinking diarrhoea, sometimes even bloody; in ruminants, occasional painful swelling of the inferior abdominal region with formation of abscesses and of a fistula of the abomasum, or even prolapse (fistula very rarely occurs in the second stomach). These symptoms are complicated by disturbance of the nervous system: paralysis (*tabes arsenicalis*), tottering, trembling, weakness, stupor, enlarged pupils, etc. Pulse is feeble or imperceptible and very rapid (weakness of the heart); bodily temperature unevenly distributed; breathing often very much accelerated and strained. Duration: a few hours to several days. On external application, as, e.g., from wounds, great swelling is noted, and on bathing, the skin is inflamed in various degrees—this in addition to the above-mentioned symptoms.

**Post-mortem Lesions.**—Dissection reveals corrosive gastro-enteritis, with redness, swelling, ecchymosis and erosion of mucous membranes, especially those of the stomach, i.e., of the abomasum. Occasionally perforation of the walls of abomasum and abdomen. Fatty degeneration of the gastric glands (*glandular gastritis, adenitis parenchymatosa*). Fatty degenerations of internal organs, such as the liver, kidneys, heart and brain. Ecchymosis, especially under the endocardium. Mummification of the carcase.

**Therapeutics.**—The principal antidote is the hydrated peroxide of iron (liquor of sulphate of iron, 100 parts mixed with 250 parts of water, to which is added 15 parts of calcined magnesia, in 250 parts of water). The hydrated oxide of iron so formed makes an insoluble compound with the arsenic. To dogs give of this arsenical antidote a table-spoonful every 15 minutes; to pigs and smaller ruminants several spoonfuls; to horses and cattle 10-40 ounces. A simple solution of calcined magnesia in 20 parts of water also produces an insoluble compound with the arsenic (dose of the calcined magnesia for dogs 7 to 15 grains every 15 minutes; for horses 1 to 3 drams; and for cattle 2 to 6 drams). Other antidotes are sulphur, sulphate of potash and sulphate of iron. If these remedies are not at hand, use any preparation of iron, such as the familiar household specific, water in which red-hot iron has been cooled, or lime-water, sugar-water, the white of eggs and mucilaginous remedies. Oil must be carefully avoided.

(B.)—CHRONIC ARSENICAL POISONING IN CATTLE CAUSED BY BREATHING ARSENICAL FUMES.

**Symptoms.**—Chronic diarrhoea and cough. A hidebound condition of the skin, with epidermal desquamation (chronic eczema squamosum), muscular atrophy beginning at the hind quarters, paralytic weakness, also starting from behind, increasing loss of flesh, and cachexia. Cessation of milk, abortion, delay in passing the after-birth, difficulty in conception.

**Post-mortem Lesions.**—Great emaciation of the carcase, general hydramia; sloughs, ulcers and scars in the abomasum, less frequently in the first stomach; red spots, inflammation of mucous membranes in abomasum and small intestine; ulcers on the membrane of trachea. Tubercular, caseous broncho-pneumonia occurs as a secondary lesion.

**Therapeutics.**—Prophylaxis, i.e., cease feeding with fodder covered with the deposited chemical dust; treat diarrhoea, cough, loss of flesh, weakness, &c., according to symptoms.

The so-called "chemical-fumes sickness" (Hüttenrauchkrankheit) in the neighbourhood of the Freiberg works must, according to Haubner's description, be regarded as essentially a chronic arsenical poisoning. The fumes consist mainly of sulphurous and arsenical acids, with smaller quantities of lead and zinc vapours and traces of earthy matter. The lead and zinc cannot, as Haubner proved, be regarded as the poisonous agents. Sulphurous acid is not present in the fine dust which settles upon and covers all the neighbouring vegetation, and which must be regarded as the exciting cause of the sickness in question, and which consequently must be arsenical poisoning, since arsenic is found in the organs of all animals affected. The so-called "caseous pneumonia caused by fumes from the works," which Haubner and Siedamgrotzky classified as a peculiar inhalation-pneumonia, has been shown by Johne's investigations to be genuine tuberculous broncho-pneumonia. Nevertheless, the inhalation of the fine dust by the animals when eating the fodder on which it has fallen is of great importance as a pre-disposing cause, which facilitates infection by the bacilli of tuberculosis. The consumption of this dust, when the amount of arsenic is larger, may even lead to acute arsenical poisoning (see previous paragraph). The so-called "sour or acid-sickness," which Häubner attributed to the sulphur in the fumes (and not to the dust), and which took the form of anaemia, with brittleness of the bones, has nothing in common with the above-named disease caused by eating fodder covered with arsenic-containing dust (chronic arsenical poisoning).

LEAD-POISONING (*Saturnism, Plumbism*).

1. **Symptoms of Acute Lead Poisoning** are at first those of gastro-enteritis: vomiting, salivation, restlessness, bowel-pains, continuous constipation; in ruminants, tympanites, later (at times), stinking diarrhoea and suppression of the secretions (urine, milk). To these supervene nervous disorders, convulsive movements, especially of head and neck, trembling (tremor saturninus), spasms of the jaw, attacks resembling epilepsy, intermittent irritability, even mania (mania saturnina), convulsions and spasms like those of chorea. Then also there appear weakness, somnolence, partial or complete paralysis, particularly of the hind-legs (paralysis saturnina), and insensibility to pain (anaesthesia saturnina). The pulse is hard and thready, often slow, and the breathing difficult and even dyspnoeic.

2. **Symptoms of Chronic Lead Poisoning**.—Increasing emaciation and weakness (tabes and cachexia saturnina), constipation, with colic, affected locomotion (arthralgia saturnina and rheumatismus saturnina), brain disorders as in the acute form (encephalopathia saturnina), especially epileptic fits (eclampsia saturnina); in horses, whistling or roaring; in goats, abortion and sterility. Moreover, there is development of ulcerous stomatitis (blue line on the gums), muscular atrophy and permanent muscular contraction (atrophia and contractura saturnina), and of chronic nephritis (contracted kidney).

**Post-mortem Appearances**.—Paleness and contraction of the intestinal canal; in chronic cases, thickening of the mucous membrane of the digestive apparatus, localised redness, inflammation, formation of scabs and of ulcers, with metallic discolouration of the mucous membrane from lead-grey to deep black. Hydrocephalus and hydrorrhachis, internal and external; contraction of the kidneys. In cases of death resembling apoplexy no essential changes are found.

**Therapeutics**.—Administration of sulphuric acid in mucilage or in water, as well as sulphates of sodium, potassium and magnesium, in order to produce insoluble sulphate of lead. Emetics and purgatives. Albumen, milk, mucilage. Intern-

ally, iodide of potassium (which hastens the removal of the lead, as iodide of lead). Administer symptomatically—morphia, atropine, nitrite of amyl, chloral hydrate, etc., to prevent lead colic, eclampsia, etc. In horses resort to tracheotomy.

#### MERCURIAL POISONING (*Mercurialism*)

**Symptoms.**—The phenomena of genuine mercurial poisoning are as follows, apart from the local corrosive action of the several preparations :

1. Salivation, ulcerous stomatitis, loosening of the flesh of the gums, loosening and even loss of the teeth in ruminants, bad-smelling mouth.
2. Gastro-intestinal catarrh ; later, profuse and even bloody diarrhoea, with greyish green, liquid, foetid faeces, smelling sometimes like putrid flesh.
3. Cough, putrescent nasal discharge, broncho-blennorrhoea and even broncho-pneumonia, strained and even dyspnoeic breathing, badly-smelling breath, groaning and bleeding at the nose.
4. Eczema of the skin, chiefly impetiginous and squamous, beginning with excessive itching, which leads to gnawing and rubbing, loss of hair, weeping, formation of pus and scabs, together with considerable thickening of the skin and swelling of the subcutis ; also growth of vesicles and pustules, the latter, however, without a depression. Most frequent location is where the ointment has been rubbed in, round the eyes, the mouth, the anus, the udder, the vagina and the creases about the joints.
5. Weakness resembling paralysis, great apathy, stupor, drowsiness, trembling (tremor mercurialis), timidity and delirium (erithismus mercurialis). Also paralysis of single muscles, amaurosis, deafness, anesthesia and emaciation. Death often occurs in coma, and rarely in convulsions.
6. Temperature normal, or occasionally raised, but the pulse, on the contrary, much accelerated and feeble, haemorrhage in different organs, especially in the mucous membranes of the nose, lungs, bowels, and uterus (abortion). Death follows sometimes from internal haemorrhage.
7. Aneurism as the result of nephritis and incrustation of lime in the kidneys, often lasting several days, cylindruria, albuminuria.

**Course of the Disease.**—This is very rapid in young animals when there is great local corrosion of stomach and bowels (sublimate may produce fatal results in an hour, without the stage of general mercurialism being reached). The duration of the acute form varies from 10 to 14 days. It may, however, be chronic, lasting several weeks, or even months. Generally, the symptomatic appearances are not so complete as described above.

**Post-mortem Appearances.**—The skin and subcutaneous tissue is found to be anaemic, the latter infiltrated with serum and blood-stained fluid under the eczematous parts of the skin. The muscular apparatus is exceedingly pale, flabby as if boiled, permeated with spotted, dark-brownish-red ecchymoses, and at the same time largely infiltrated with a gelatinous fluid. The blood appears dark-red, greasy and badly coagulated. Inflammatory signs are present in different degrees upon the mucous membrane of the digestive tract, such as ulcerous stomatitis (this may also be absent), dots and spots of red, haemorrhagic erosions, ulcers in the stomach, especially on the top of the prominences of the mucous membrane and in the abomasum; the mucosa is often oedematous and swollen, the submucosa infiltrated with serum, so that the mucous membrane of the bowels forms loose folds. Excessive intestinal anaemia is also observed. The sub-peritoneal connective tissue is likewise oedematous and infiltrated with spotted haemorrhages. The liver is anaemic and swollen; the kidneys are infiltrated with serum, anaemic, and ecchymosed; sometimes they are completely incrusted with lime; the epithelium of the kidneys is in a condition of fatty-degeneration. In addition there are also sometimes rhinitis, laryngitis, tracheitis, catarrhal bronchitis and even croup of the larynx and trachea. The lungs are full of blood, infiltrated with haemorrhagic and broncho-pneumonic foci, or even with abscesses, the bronchial and mediastinal glands are swollen and hyperæmic; subpleural ecchymosis is also present. Finally, myocarditic degeneration and haemorrhage are found between the muscular fibres of the heart, beneath the epicardium and endocardium; subarachnoidal infiltration of blood, with anaemic, soft brain-substance, gleaming like water, and dotted haemorrhages on the superficial layer of the brain (cortex).

**Therapeutics of General Mercurial Poisoning.**—Administration of sulphur compounds, so as to form an

insoluble compound of the mercury and sulphur, is recommended, viz., flowers of sulphur, potassium sulphate, etc. Also iron filings, and sulphate of iron; albumen in solution; and iodide of potassium. No common salt. In addition to these, adopt symptomatic treatment for gastro-enteritis, stomatitis, bronchitis, eczema, etc.

#### POISONING BY COPPER (*Cuprism*).

**Symptoms.**—They are those of gastro-enteritis, viz., vomiting greenish matter, loss of appetite, colic and diarrhoea. Later follow great weakness, trembling, convulsions, anaesthesia; small, thready, slow pulse, quickened breathing.—*Autopsy* reveals the signs of acute gastro-enteritis. According to Trasböt, considerable enlargement of the stomach is observed.—*Therapeutics*: albumen, mucilage, milk, reduced iron, iron filings, sulphur, calcined magnesia, ferrocyanide of potash. According to symptoms administer narcotics.

#### POISONING BY ZINC.

**Symptoms.**—Vomiting, colic, diarrhoea, weakness and signs of paralysis; if long continued, anaemia and cachexia.—*Post-mortem* shows shrinking and anaemia of the gastrointestinal mucous membrane, with gastro-enteritis in the form of localised inflammatory foci and erosions.—*Therapeutics*: albumen, mucilage, sugar, water, sulphur and calcined magnesia.

#### POISONING BY TARTAR EMETIC.

**Symptoms.**—Similar to those of poisoning by corrosive alkalis and acids: ulcerous stomatitis with salivation, vomiting (in isolated cases also in horses), colic, diarrhoea, dizziness, trembling, spasms, paralysis, small, hard and often imperceptible pulse, accelerated breathing, even pneumonia; rapid death.—*Autopsy* shows, inflammation and erosion of the mucous membranes of the entire digestive tract, especially the stomach (pharyngitis, stomatitis, oesphagitis, gastritis and ulcerous enteritis); also hyperæmia of the lungs, scaling or peeling of the bronchial epithelium, haemorrhagic congestion, and even inflammation of the lungs. According to Trasböt, en-

## *POISONING BY COMMON SALT AND BRINE.* 187

largement of the stomach is also a characteristic symptom of this form of poisoning.—*Therapeutics*: first of all, remedies containing tannin (decoction of oak-bark, ink, tannin); mucilaginous and oily substances; narcotics, especially opium; with dogs, small pieces of ice (ice-pills).

## *POISONING BY COMMON SALT, BRINE, AND THE BRINE OF HERRINGS AND MEAT.*

**Symptoms.**—In cases of poisoning by *common salt*, we have chiefly signs of gastro-enteritis, combined with conditions resembling paralysis. Cessation of eating, a dry, hot, red mouth, great thirst, vomiting, colic pains, watery diarrhoea, and frequent urination, are the signs first seen. Then come great bodily weakness, staggering, falling, paralysis of varying distribution (particularly of the hind limbs in horses), stupor, dilation of the pupils, even blindness, and tremblings; in pigs (but less frequently in horses and dogs), now and then fits of cramp resembling epilepsy; weakness of the heart, small, weak pulse, dyspnoea. Death occurs within six to forty-eight hours. Should the sickness last longer, a condition of anaemia sets in. In cattle, the appearances of enteritis sometimes manifest themselves.

2. In *poisoning by herring brine* the symptoms of ptomaine poisoning are added to the above (brain irritation and spasms). Especially characteristic signs are: grinding the teeth, spasms of the jaw and cramps of epileptic nature, opisthotonus and pleurostethotonus, rolling the eyes (*nystagmus*), spasmodic blinking, widespread twitching, twisting the body round, squatting on the haunches, phenomena of chronic hydrocephalus, stupor, insensibility, amaurotic pupils and blindness. In cows, abortion, even prolapse of the uterus. Course very acute, death generally occurring after six to twelve hours.

**Post-mortem Lesions** are those of acute gastro-enteritis, the abomasum in ruminants being especially affected. Also conspicuously bright-red blood, very fluid, with ecchymoses in various organs. The membrane of the bladder is often reddened. Inflammatory changes are not unfrequently absent in the intestinal canal, especially in brine poisoning; on the other hand, one generally finds in the latter hyperæmia of the brain with internal and external hydrocephalus.

**Therapeutics.**—Plentiful supply of water, mucilaginous

and oily remedies, stimulants (ether subcutaneously, camphor, wine, etc.). Where the excitement is great narcotics may be given.

#### POISONING WITH SALTPIPETRE (NITRATE OF SODA AND NITRATE OF POTASH).

**Symptoms** are those of gastro-enteritis combined with paralysis; colic, diarrhoea, tympanites (in cattle), vomiting, slavering and choking movements; also fall of bodily temperature, prostration, tottering, falling, stupor, tremblings, palpitating heart-beat and polyuria. Death occasionally takes place from apoplexy; more rarely is accompanied by spasms, rolling of the eyes, etc. The course is commonly acute, death occurring indeed often in five minutes. Duration: mostly from half an hour to several hours, rarely more than twelve. *Autopsy* reveals: haemorrhagic gastro-enteritis, especially in the stomach (abomasum) and small intestine; purple to cherry-brown red discolouration and ulcerous degeneration of the mucous membrane; contents of stomach, a brownish-red fluid; hyperæmia of the abdominal viscera, inflammation and ecchymoses of the kidneys and mucous membrane of the urinary bladder; blood fluid and bright red or discoloured.—*Therapeutics*: emollients, narcotics or stimulants (camphor, coffee, ether, etc.).

#### POISONING BY GLAUBER SALTS, EPSOM SALTS AND SALTS USED AS MANURES.

**Symptoms**.—Colic, intense thirst, watery diarrhoea, suppressed peristaltes, paralytic condition, inability to stand up, increasing debility; death after several days. Similar appearances are noted to those of poisoning by kainite and super-phosphates.—*Treatment*: as in cases of poisoning by common salt.

#### POISONING BY CORROSIVE ALKALIS (CAUSTIC POTASH, CAUSTIC SODA, SPIRIT OF AMMONIA).

**Symptoms**.—Erosion of the lips and mucous membrane of mouth, tongue and pharynx; foaming, difficulty of swallowing, colic. When ammonia is the poison taken, coughing also, difficult breathing, and the coughing-up of skinny, croup-like matter. Muscular weakness and collapse. *Autopsy* reveals:

croupy stomatitis and pharyngitis. In ammonia poisoning also croupy laryngitis, tracheitis, bronchitis and pneumonia; intense inflammation, brown to reddish-black discolouration, gelatinous swelling and extensive corrosion of the stomach and intestinal membranes.—*Therapeutics*: diluted acids, especially (as household remedy) vinegar; mucilaginous and oily drinks with opium. To prevent collapse, use camphor and ether. Emetics and purgatives are contra-indicated.

#### POISONING BY CORROSIVE ACIDS (*Sulphuric and Sulphurous*).

**Symptoms.**—Those of sulphuric acid poisoning are ulcerous stomatitis and pharyngitis, with violent gastro-enteritis, swollen and bleeding lips, eroded and much-inflamed mouth, slavering, difficulty of swallowing, violent colic pains, completely suppressed peristalsis, rapid and feeble pulse. Speedy death.

In poisoning by sulphurous acid (inhalation), one finds inflammation of the mucous membrane of the breathing apparatus, which varies much in intensity; œdema of the lungs, with inflammation and emphysema.—*Autopsy* shows (in sulphuric acid poisoning), corrosion of the mucous membranes from the lips to the bowels, stomatitis, glossitis, pharyngitis, œsophagitis, gastritis and enteritis.—*Treatment*: diluted alkalis, soap-suds, lime water, dilute solutions of soda, potash and burnt magnesia. Opium along with mucilaginous and oily remedies; stimulants (camphor, ether).

#### POISONING BY VINEGAR.

**Symptoms.**—Loss of appetite, colic, diarrhoea, staggering, stupor, twitchings, collapse, quickening of respirations, and pulse.—*Treatment*: alkalis, stimulants.

#### POISONING BY CARBOLIC ACID.

**Symptoms.**—Diminished appetite, slavering, vomiting, diarrhoea, slight colic pains, arching of the back; urine dirty, muddy, greenish-brown, containing albumen and smelling of carbolic acid. Paresis, and paralysis of hind legs, or paralysis of the entire body, sometimes sudden falling down, tremblings, timidity, restlessness, continuous convulsive spasms, stupor, coma, collapse. Fall of internal temperature with acceleration

of pulse. Irregular and difficult breathing. Sometimes the symptoms of nephritis (white and red blood corpuscles, casts in the urine). Local corrosion when the acid has been concentrated. *Autopsy* shows : haemorrhagic gastro-enteritis, fatty degeneration of the liver and kidneys, parenchymatous degeneration of the heart, and even parenchymatous nephritis. Dark, badly coagulated and greasy blood. Hyperæmia of brain and lungs, pia-oedema, serous discharge into the ventricles and a smell of carbolic acid in all the organs. Where locally applied, corrosion.—*Treatment* Sulphates, and particularly Glauber's salt, which neutralizes the acid. Soap, lime-water. Stimulants : ether, camphor, spirits of wine (alcohol), coffee, wine.

#### POISONING BY IODOFORM.

**Symptoms.**—1. The *acute form* of poisoning shows itself first in slight gastric disturbances (loss of appetite, vomiting and constipation), then in sleepiness, stupor and coma, which are interrupted by fits of spasms. Dogs show sometimes at the beginning great excitement and even mania. Later one finds a great fall of internal temperature, and signs of heart weakness are developed (very frequent, feeble pulse, palpitation, dyspnoea, oliguria and albuminuria).

2. The *chronic form* of poisoning manifests the symptoms of iodism. They consist in emaciation, atrophy of the glands, especially the lacteal gland, iodine-eczema and catarrh of the mucous membranes, particularly those of the nose (iodine catarrh), of the conjunctiva and of the mucous membranes of larynx and bronchi.—*Autopsy* reveals : fatty degeneration of the larger glands of the body (liver, kidneys), as the most important change, also of the muscles of the heart and skeleton, with occasional glomerulo-nephritis.—*Therapeutics* : It is, above all, imperative to remove the poison by the use of emetics, if this can be done; we have, however, no direct antidote. One may attempt to render it ineffective by administering strong doses of starch flour. The treatment is, however, chiefly symptomatic and stimulative.

**Chlorine Poisoning.**—The symptoms in this consist of severe inflammation of the mucous membrane of the upper air-passages, in coughing and extreme difficulty of breathing, with attacks of choking. Rost describes a case of a horse being poisoned in a chemical works, by being lodged near the chlorine chamber when open.

**Petroleum Poisoning.**—*Symptoms*: Gastric disturbance, dizziness, stupor, appearance of intoxication, paralysis. The treatment consists in the administration of emetics and stimulants.

## II.—VEGETABLE POISONS.

## LUPINOSIS OF SHEEP.

**Historico-statistic Notes.**—Isolated cases of illness from eating lupine were already noted by Wienands, Liebscher, and Gütlich in the early sixties of the nineteenth century. But the first records of widespread sickness date from 1872. From this point, and especially since 1875, the affection of large numbers of animals became much commoner, so that the Prussian Ministry of Agriculture found it necessary to instruct the veterinary colleges of Hanover and Berlin to enquire into the nature of the sickness. It was found to be most prevalent throughout the northern provinces of Germany, where the soil was specially adapted for the growth of lupine. In South Germany the disease was almost unknown. The injury inflicted upon agriculturists by lupinosis is very considerable, for frequently from half to three-quarters of the affected animals perish. In the province of Pomerania the losses amounted to many thousands a year. V. Below-Saleske reported that one district of the province lost in one year 14,138 out of a total of 240,000 sheep, besides being able to rear 13,000 fewer lambs than the usual average.

In addition to sheep, goats, horses, cattle, pigs and fallow-deer are subject to the disease. It can be transmitted to dogs, but not to rabbits.

**Etiology.**—Although similar symptoms had been noticed where no lupine had been eaten, yet true lupinosis only occurs where the plant is grown and used as fodder. It is especially the cultivated yellow lupine which is injurious, much less frequently that which bears blue or white flowers. Race, sex and age have no influence in predisposing to the disease; if lambs and ewes frequently suffer more severely than rams, the reason lies solely in their more delicate organisation. Various opinions have been uttered as to the exciting cause of the disease, but it is most probable that the injurious substance in lupine is a purely chemical poison. Evidence of this is found in the fact that the upper layers of stacks built in the open are found to be quite innocuous, the poisonous element having been washed out

by the rain. A further proof is that the degree of illness is always in direct proportion to the amount of the food consumed, and that acute or chronic symptoms may be produced according as more or less of the poisonous extract is given ; and lastly, that it is possible to extract the matter which produces lupinosis by washing the lupine in water containing 2-3 per cent. of caustic soda. Kühn suggested "Ictrogon" as the name of this extracted chemical matter. Arnold and Schneidemühl call it "Lupinotoxin," which is certainly a more appropriate name than Kühn's.

**Properties and Preparation of Lupinotoxin.**—It is only slightly soluble in water, but more easily in water made somewhat alkaline. Dry heat does not destroy the poison, even if raised to boiling point and maintained for three hours. On the other hand, steaming with 1-1½ atmospheres of high pressure for several hours very much weakens it, and if the lupine be steamed with fully two atmospheres of high pressure the poison is entirely destroyed.

This poison lies chiefly in the pods, seeds and husks of the plant, but is also present in all parts. Arnold and Schneidemühl propose the following method of extraction :—Finely-powdered lupines are mixed with a 1½ per cent. solution of soda, heated to 105°-120° F., and stirred to a consistency of thick broth. After standing two days the liquid is pressed out. The filtrate is then evaporated in a water bath (140° F.), mixed with acetic acid, filtered and treated with a solution of acetate of lead the filtrate saturated with sulphuretted hydrogen, evaporated at 120° F., and poured into 15 times its volume of 98° alcohol. The deposit is collected after 24 hours, dried and dissolved in water (it is a brown, resinous substance), mixed with acetate of lead and ammonia, and after precipitation, washed with distilled water containing ammonia, alcohol and ether, suspended in water and decomposed by sulphuretted hydrogen ; the precipitate is then evaporated at 160° F. and added to ten times its volume of 98 per cent. alcohol.

Respecting the generation of this poison in the lupine plant nothing certain is known. The possibility of its development as a result of metabolism by parasitic fungi on the lupines is, however, universally admitted.

**Post-mortem Lesions.**—The principal changes detected on dissection of sheep suffering from this form of poisoning consist in parenchymatous inflammation of the internal organs, especially of liver and kidneys, with appearances of jaundice and a gradually developing haemorrhagic diathesis.

I. The *liver* affection may be characterised as an acute parenchymatous hepatitis. A high degree of granular, albuminous opacity of the liver-cells is found, whereby the liver may be abnormally enlarged, or of its usual size, according as it has been previously affected by fatty infiltration. This initial granular opacity is quickly followed by fatty degeneration of

the cells of the liver, which grow larger and contain larger or smaller drops of fat in addition to the granules of albumen. The intensity of this degeneration seems to depend upon the degree of nourishment of the individual. The liver becomes flabby, soft and friable. This stage of softening (at which most animals perish in a few days) is followed by atrophy (acute yellow atrophy of the liver), during which the softened fluid contents of the liver-cells are re-absorbed, and the liver itself grows much smaller. The stage of atrophy lasts about fourteen days. In chronic lupinosis these liver changes take the form of a chronic interstitial hepatitis, with new growth of interstitial connective tissue. Here the liver is smaller and firmer, sometimes lobulated and uneven, and the well-known phenomena of congestion are manifest (ascites, oedematous swelling of the spleen and intestinal mucous membrane).

2. *Jaundice*, which is generally present, affects first of all the liver, which is sometimes pale or citron-yellow, sometimes red or the colour of yellow ochre. The jaundice is hepatogenous, and produced by the enlargement of the liver and consequent biliary congestion between the cells, as well as by catarrh of the larger and finer bile-ducts. The liver may also show the discolouration of jaundice in isolated spots. In addition to the liver, the subcutis, the skin of the abdomen, the omentum and mesentery are also yellow. The gall bladder is often enormously distended and filled with bile, its mucous membrane being reddened and tumefied.

3. The *kidneys* display similar parenchymatous changes to the liver, but in lesser degree. Here, also, we find the signs of parenchymatous nephritis, consisting of granular opacity in the epithelium of the spiral tubes, accumulation of casts of exuded matter in Henle's loops, etc. In the urinary bladder, which is empty or nearly so, we find catarrhal cystitis.

4. In the *digestive organs* one notices, in addition to the jaundiced colour of the mucous membrane, a catarrhal and sometimes parenchymatous (glandular) gastritis, especially marked in the abomasum, with inflammatory redness and haemorrhage in the small intestine, along with catarrhal affection of the entire intestinal canal.

5. The *heart muscle* is very pale and tender ; the blood within the heart and in the larger veins of the body is dark and thick, and coagulates quickly on exposure to air, turning light-red. In most of the organs (bowels, skin, subcutis, peritoneum, uterus and cerebral membrane) are found capillary haemorrhages.

6. The carcase is generally much emaciated. The muscles greyish-yellow in colour, their fibres granular and in a state of fatty-degeneration, and sometimes having lost their transverse striation. The carcase decomposes quickly.

**Symptoms.**—The first sign of lupinosis is loss of appetite. Sheep, although accustomed to lupine feeding, take the plant reluctantly, and often refuse it altogether until overcome by hunger; later they refuse it altogether. Among the earliest symptoms is noticed a rise of temperature. This may even be noticed one day after eating poisonous lupine, and in acute cases reaches 104–105° F., or even higher. The character of the fever seems, however, to be remittent, and before death the temperature sinks considerably. The pulse is proportionately accelerated, reaching 130 a minute, or more.

Signs of jaundice may, in many cases, be noticed on the second or third day; in milder cases not until the fifth or sixth. They appear suddenly or gradually, and, as a rule, first in the eye. They may, however, be absent, and even the yellowish discolouration of conjunctiva has not unfrequently been missed by observers, from which one may conclude that jaundice is not necessarily a symptom of lupinosis.

As the disease advances, and indeed, sometimes from the very beginning, appearances of weakness and heaviness of the head also show themselves in the animals; they manifest stiffness of gait, rise painfully to their feet, lie about and allow themselves to be freely handled. The heaviness of the head occasionally increases to insensibility: the animals hang down their heads, lean against the nearest support, or lie stretched at full length. They walk or stagger sideways, or backwards and forwards. They grind the teeth. The lower jaw is constantly agitated and the jaw muscles spasmodically contracted. Trismus frequently occurs, and they shrink with fear at the slightest noise.

Defaecation is at first usually retarded, the dung is hard and covered with yellow slime; later, it often becomes like tar and dark-brown, owing to admixture of blood. Now and then diarrhoea occurs. The urine, which is passed in small quantities, but frequently, is yellow in colour, containing mostly biliary pigment, and invariably bile-acids and albumen. Besides these, one finds it to contain epithelial cells of the kidneys and bladder, round cells, as well as cylindrical urinary casts of various kinds.

Respiration is at first regular, but later becomes more rapid and painful (100 per minute). In some sheep a mucous discharge from the nose was observed, as well as blood-stained foam.

A fatal end may follow in one to two days, but the usual duration of the sickness is four to five days. Death is caused by great emaciation and rapid failure of strength. Should the ailment tend towards improvement and recovery, the symptoms may pass away more or less quickly. But at times the apparent improvement develops into cachexia.

The *prognosis* of lupinosis is very unfavourable. Only when the toxic effect is slight, and the offending fodder is promptly discontinued, is there any hope of recovery. Acute cases are generally fatal.

**Chronic Lupinosis.**—This arises from persistent feeding with only slightly poisonous lupine, and generally runs its course without jaundice under the aspect of chlorosis and cachexia. But nasal catarrh, conjunctivitis and, in solitary cases, inflammation of the skin and formation of scabs on the lips and ears have been noticed. Zurn also reports swellings on certain parts of the head (eyelids, lips, ears), with exudation of a yellowish fluid and subsequent formation of scabs.

**Treatment** must be prophylactic, for up to the present no specific antidote to the disease itself is known. If, from economic reasons, it be impossible to entirely exclude lupines from the food, it is advisable to find out by experiment which fields produce the poisonous quality, taking each field separately, so as to avoid mistakes. This may be done as follows :—

1. Use the suspected lupine along with an excess of sound fodder, say, in the proportion of 1 to 6 or 10, and, if possible, for older animals only.
2. Allow the lupine-hay to be washed through in small heaps by rain in the open, and from larger heaps use only the upper layers for food.
3. Soak the injurious lupine in water, or still better, in a 1 per cent. solution of soda, for 48 hours, renewing the liquid from time to time.
4. Steam it for several hours under a pressure of two atmospheres.
5. Or place the lupine in thrice its bulk of water, to which 4 quarts of ammonia spirit have been added for each cwt. of lupine, and let the mixture stand two or three days at about 55° F. Afterwards soak in water for seven to ten days.

If the sickness has already broken out, avoid the use of all alkaline mixtures, especially alkaline drinking water. It will be wise to add a little acid to the water, as it renders the lupine-poison insoluble. Furthermore, we recommend the use of evacuants, in order to remove the poison as rapidly as possible; but avoid Glauber's salt, which hastens solution, using rather oily remedies and the yeast of beer. The flesh of animals not too severely affected is quite fit for food.

**Lupinosis in Horses.**—As regards its anatomical changes and general symptoms this is similar to the same condition in sheep. It usually arises from the admixture of lupine, either in the fodder or litter. There is usually a more violent inflammation of the intestinal canal than is the case with sheep, and the first symptoms shown are sudden refusal of food, even of the best oats or meadow hay, abstinence from drink, dulness of the sensorium which may amount to extreme cerebral depression, and during which the animal at times lays its head on the manger, grinds its teeth, hangs back with drooping head and displays an unsteady, staggering gait. The usual accompaniment of low fever may also become acute, the pulse rising to 60 a minute, or more. Breathing is generally steady, but may also be accelerated to 36—40 per minute. More or less intense icterus (jaundice) is present (conjunctiva being orange-red to citron-yellow, mucous membranes of mouth, nose and vagina clayey yellow). The tongue shows a coating of yellow mucus; evacuation becomes infrequent, faeces in small balls, often covered with mucus and very foul in smell. Urination, on the contrary, is frequent, but in small quantities. After these conditions have lasted pretty evenly for about eight days, an improvement sets in, and recovery follows in from 16—21 days. Fatal ending is unknown. Among unusual symptoms must also be mentioned: slight attacks of colic, an orange-red and ropy nasal discharge, necrotic patches on the mucous membrane of the tongue about the size of a crown-piece, drying up of the skin over the bridge of the nose, skin inflammation accompanied by yellow, lustrous exudation, formation of crusts and detachment of the epidermis on the under lip and on the pasterns, with simultaneous edematous swellings as far as the carpal and tarsal joints.

**Supplement to Lupinosis.**—The characteristic signs of this complaint greatly resemble those of other, as yet unexplained, diseases, which have no connection with it. The most striking likeness is that between lupinosis and the so-called "typhous inflammation of the liver," or "malignant jaundice" of sheep after feeding with a large quantity of potato mash. The impression gained is that of complete identity between the two diseases. The complaint described by Sander as "liver-typhus in horses," and traced to feeding in inundated pastures, reminds one in all its symptoms of acute lupinosis. Reinemann and Jansen found that, under certain circumstances,

eating of the straw of peas, beans and vetch may produce conditions like those of lupinosis. Stöhr noticed the same pathological changes in horses after feeding on vetch as in chronic lupinosis among sheep. In oxen, a dry necrosis of round patches on the skin was noted, and in horses and pigs, bowel inflammation. Wenke reports even fatal cases in horses after feeding on vetch (symptoms of paralysis).

The aspect of chronic lupinosis reminds one involuntarily of the so-called "Schweinsberger sickness," or "induration of the liver" in horses. (See chapter on liver diseases). Finally, there is so indisputable a resemblance between lupinosis, both acute and chronic, and the so-called clover-disease of horses, that we consider it advisable to discuss the latter at this point.

#### THE SO-CALLED CLOVER DISEASE.

**Etiology.**—The injurious effects of an exclusive diet of clover for horses, and sometimes for cattle, arise, according to the unanimous opinion of authors, only from the use of bastard or Swedish clover (*trifolium hybridum*). The pathogenic properties of the latter have long been known. The true source of the trouble is probably to be sought in the mould (*Uromyces apiculatus*?), which appears to produce the symptoms detailed below, both by acute irritation of the gastro-intestinal mucous membranes, and also, indirectly, by its effect upon the brain and liver, after absorption of the toxic products of metabolism.

**Symptoms.**—These vary according as the skin and oral mucous membrane, or the internal organs be attacked. The changes consist, firstly, of swelling of the fore part of the head, with excessive stomatitis, during which ulcers are formed within and without the cleft of the lips, as well as exudations from the epithelium of the tongue. Similar changes have been noticed on such parts of head or limbs as are unpigmented. Here yellowish patches are formed, in some cases covered with blisters, which, on drying gradually, peel off and are very painful (similar appearances are noticed in lupinosis). Other general symptoms resembling those of lupinosis are yellow discoloration (like jaundice) of the oral mucous membrane and conjunctiva, attacks of colic, great faintness and somnolence, along with nervous excitability (convulsions, raving, diarrhoea and attacks like those of epilepsy), staggering, paraplegia, amaurosis, paralysis of the

pharynx. The course of the paraplegia, which is frequently ensoëtic, closely resembles sub-acute inflammation of the brain, and is usually rapid and fatal.—Remedies are seldom of any avail. The chief thing to do is to discontinue the offending clover. Other therapeutic remedies are merely symptomatic.

#### POISONING BY SNAKE-GRASS (*Equisetum palustre*), LIMOSUM, ARVENSE AND HIEMALE. THE SO-CALLED STAGGERING SICKNESS.

**Symptoms.**—At first, excitement and anxiety, the sensorium remaining unaffected; later, uncertainty of movement, reeling and staggering; at last, paralysis of hinder limbs, tumbling down, general paralysis, insensibility to external irritants, unconsciousness and coma. Pulse accelerated, appetite at first normal, but in course of time great disturbance of nutrition; sugar in the urine. Course sometimes very acute, death occurring in a few hours, but sometimes protracted (two to eight days), and at times chronic (one to several weeks). In cattle, after excessive eating continuous diarrhoea becomes a prominent characteristic along with the paralytic symptoms; while, if the food be persisted with, cachexia and hydremia, combined with weakness bordering on paralysis, make their appearance.—*Autopsy* reveals: hyperemia, œœma, dropsical effusions on the brain and spinal cord, especially on the cerebellum; in cases of longer duration, hydremia. Sometimes inflammatory changes in the mucous membrane of stomach and bowels.—*Therapeutics*: change of fodder, purgatives and stimulants, especially camphor; blisters along the spine.

#### POISONING BY BEECH-NUT OIL-CAKES.

**Symptoms.**—Very violent colic (intestinal tetanus), mania, fits of madness, continuous convulsive spasms which resemble those of strychnine tetanus, signs of paralysis, staggering, tumbling, with inability to rise. Course very acute, death ensuing in a few hours from suffocation.—*Autopsy* reveals conditions as in strychnine poisoning, sometimes red spots in the intestines.—*Therapeutics*: tannin, chloral hydrate, morphia.

#### POISONING BY CASTOR-OIL SEED-CAKES.

**Symptoms.**—At first gastric appearances: vomiting, difficulty of swallowing, colic, bloody diarrhoea and anuria.

## POISONING BY COLCHICUM AUTUMNALE. 199

Then weakness in the hind limbs, inability to stand, dulness of the sensorium.—*Autopsy* shows hemorrhagic gastro-enteritis and sometimes also nephritis.—*Treatment* consists in administration of mucilaginous remedies, opium, tannin and stimulants.

## POISONING BY COTTON-SEED MEAL.

**Symptoms.**—Only young animals are usually affected. They display disorder of digestive and urinary apparatus : tympanites, diarrhoea (afterwards bloody), hemorrhage from the urinary organs, albuminuria, strangury and paralysis of the bladder. Duration is variable.—*Autopsy* reveals in the acute form hemorrhagic gastro-enteritis, opaque swelling of liver and kidneys, oedema of the lungs, dark-red urine, collection of liquid in the cavities of the body. There is no enlargement of the spleen. In chronic cases only, one finds general emaciation and dropsical phenomena, but at times, also parenchymatous nephritis.—*Therapeutics* : change of food, doses of castor-oil (for calves, 2-4 oz. ; for lambs, 2-6 drams). Other treatment according to symptoms.

## POISONING BY COLCHICUM AUTUMNALE (*Meadow Saffron*).

**Symptoms.**—At first those of acute gastro-enteritis : loss of appetite and vomiting, colic, violent and sometimes bloody and dysenteric diarrhoea, tympanites in cows, sometimes difficulty in swallowing. Increased urination, the fluid often being deep to dark-red (haematuria) ; later, when a nephritis has formed, anuria sets in. Dulness of the sensorium, apathy and somnolence to the extent of insensibility. Weakness, especially in hinder parts ; staggering, tumbling, inability to rise, tremblings. Small and, at times, imperceptible pulse ; violent palpitation in horses ; coldness of the extremities, sweating, livid discolouration of the visible mucous membranes, dilation of the pupils and accelerated respiration. Death occurs in from one to three days.—*Autopsy* reveals hemorrhagic gastro-enteritis. Widespread ecchymosis, dark, thick and badly coagulated blood. Fatty degeneration of the liver (in one case on record).—*Therapeutics* : tannin, or remedies containing it, opium, mucilaginous drinks. In cattle, rumenotomy. Treat the gastro-enteritis and paralysis according to symptoms.

POISONING BY WILD POPPIES (*Papaver Rhazæs*).

**Symptoms.**—Slavering, colic, constipation, tympanites, diarrhoea, intestinal bleeding ; in cattle, raging fits of fury ; bellowing, gnashing of teeth, desire to butt, biting, goring and wild dashing about. Also cramp resembling epilepsy, tumbling, unconsciousness, somnolence, tremblings and the appearances of drunkenness. These attacks last on an average several hours. The termination is, however, seldom fatal. Gerlach noticed also a chronic case of poisoning by wild poppies in horses after being fed with chop containing many of these flowers. The symptoms were those of chronic immobility, dulled sensation, etc.—*Autopsy* shows : gastro-enteritis, nephritis and signs of suffocation.—*Therapeutics* : tannin, cold shower bath on the head ; in the somnolent condition, stimulants : ether, camphor, etc.

POISONING BY TOBACCO (*Nicotiana*).

**Symptoms.**—Choking, vomiting, slavering, colic, tympanites, diarrhoea (intestinal tetanus) ; polyuria. Great muscular weakness, tottering, tumbling, inability to rise, paralysis and trembling of the whole body. Continuous convulsive spasms of the muscles, opisthotonus, cramp in the diaphragm, contraction of the eye-muscles, prolapse of the nictitating membrane, amaurosis, stupor and unconsciousness. The action of the heart is at first slower, but later becomes extraordinarily accelerated and very irregular, furious palpitation ; coldness of extremities ; difficult and even dyspnoëic breathing. *Course* : If the poison is applied through the skin, often very acute, death happening even within an hour ; but generally acute and ends within a day. Convalescence may last in some cases eight to fourteen days.—*Autopsy* reveals haemorrhagic gastro-enteritis (which, however, is absent when the poisoning has been through the skin) ; widespread ecchymosis of the sub-pleural and sub-peritoneal tissues, etc. ; dark, fluid blood, hyperæmia of brain and lungs.—*Therapeutics* : Tannin, or remedies containing the same (which precipitate the nicotine) ; in cases of urgency, black coffee. Stimulants. In cattle, rumenotomy.

POISONING BY YEW-TREE LEAVES (*Taxus baccata*).

**Symptoms.**—Often sudden death resembling apoplexy,

with staggering, falling and convulsions within a quarter of an hour. In other cases, the condition lasts longer (several hours to a few days), and is accompanied by gastro-enteritic symptoms : choking, foaming, vomiting, inflation, costiveness and frequent urination. Then stupor, tottering, tumbling, tremblings, spasms, the latter sometimes in the form of recurrent fits like epilepsy. Pulse small and slow ; cold extremities.—*Autopsy* shows gastro-enteritis (not constant), hyperæmia of the brain and oedema ; dark fluid blood.—*Therapeutics* : Often impossible, owing to rapidity of the course. In longer cases prescribe purgatives ; other treatment according to symptoms. In cattle, in case of urgency, rumenotomy.

#### POISONING BY CORN-COCKLE (*Agrostemma githago*).

**Symptoms.**—Phenomena of colic, vomiting, slavering, difficulty of swallowing, diarrhoea, regurgitation, paralysis, stupor, trembling, laboured breathing.—*Autopsy* shows intense gastro-enteritis, hyperæmia of brain and spinal cord, softening of the grey matter of the latter and appearances of suffocation.—*Therapeutics* : Change of fodder, purgatives, stimulants.

#### POISONING BY BOX-TREE LEAVES (*Buxus sempervirens*).

**Symptoms.**—Vomiting, colic, diarrhoea ; dizziness, stupor, appearance of being drunk, sometimes very speedy and convulsive death.—*Autopsy* reveals gastro-enteritis, with signs of asphyxia.—*Therapeutics* : Symptomatic ; tannin.

#### POISONING BY DIGITALIS (*Foxglove*).

**Symptoms.**—Slavering, retching, vomiting, colic, violent diarrhoea. Action of the heart is at first retarded, but later abnormally stimulated, palpitations, the beating of the heart gives a distinctly metallic sound. Pulse small, irregular and feeling as though it gave a double beat, finally quite imperceptible. Polyuria, albuminuria, strangury ; excitement followed by stupor, faintness, staggering, paralysis of the under-lip, contraction of the pupils, spasms.—*Autopsy* reveals gastro-enteritis, endocarditis, myocarditis ; heart dilated, appearances of suffocation (hyperæmia of the lungs and hypostasis, ecchymosis, dark, fluid blood).—*Treatment* : There is no known specific antidote, therefore this must be according to the symptoms,

partly soothing, partly stimulative. Tannin may at least be tried. Chief stimulant, camphor.

#### POISONING BY OLEANDER (*Nerium oleander*).

**Symptoms** consist of vomiting, colic, diarrhoea, polyuria, palpitation; irregular, very feeble and retarded pulse; occasionally signs of excitement are noticed. Death follows with the appearances of paralysis (weakness, muscular tremblings).—On *autopsy* gastro-enteritis is found.—*Treatment* according to symptoms.

#### POISONING BY LABURNUM (*Cytisus*).

**Symptoms** are slavering, retching, vomiting, sleepiness, staggering and general paralysis.—On *autopsy*, slight gastro-enteritis is found.—*Treatment*: Emetics, tannin; further treatment according to symptoms.

#### POISONING BY THE ERGOT OF RYE (*Ergotismus*).

**Symptoms**.—1. In acute poisoning, gastro-enteritic phenomena, which often show similarity to rinderpest; slavering, vomiting, colic, diarrhoea; ulcerous stomatitis like that of foot-and-mouth disease. Labour pains, abortion and even prolapse of the uterus with pregnant animals. Dizziness, dulness of the sensorium, insensibility to pain, paralysis, dilation of the pupils, cramp of the flexor muscles (*ergotismus spasmodicus*). 2. In chronic cases; necrosis of the extremities, such as the ears, tail, teats, claws and lower leg (*ergotismus gangrenosus*), sterility. Enzoötic abortion.—*Autopsy* reveals gastro-enteritis, signs of suffocation.—*Therapeutics*: Tannin, remedies such as will dilate the vessels, anti-spasmodic remedies; chloral hydrate and morphia.

#### POISONING BY PLANTS CONTAINING OIL OF TURPENTINE. (*The so-called Enzoötic Gastro-Enteritis.*)

**Etiology**.—Enzoötic gastro-enteritis is caused by the consumption of plants which are resinous or contain turpentine, especially the young shoots of the coniferae; and also of astringent, irritating vegetable matter containing tannin (heather, bilberry plants, gorse or branches of the elder tree). One has,

therefore, practically to deal with a case of poisoning by turpentine oil, with which all the symptoms agree. Enzoötic gastro-enteritis, first described by Chabert in 1787, is therefore clinically to be regarded as a gastro-enteritis complicated by nephritis, which arises from pasturing cattle in the forests in spring time, or when fodder is scarce, and allowing them to eat the young fir-shoots and leaves. As this particular sickness was formerly very common, as compared with the rarer instances of poisoning, and has special aspects of its own, it was described as a separate disease under the above name. Nowadays, that forest pasturage is rarer, it is better to discuss it along with the toxic forms of gastro-enteritis, among which Delafond has already classified it. As the so-called enzoötic gastro-enteritis exhibits symptoms of haematuria, it has been usually described in literature under this name, and we shall therefore be obliged to refer to it again in the chapter on "haematuria" and "haemoglobinuria." Here we propose, in the first place, to bring into prominence the gastric, or rather enteritic after-symptoms, as developed after eating matter which is resinous and contains oil of turpentine.

**Symptoms.**—This ailment, called in some places "forest or wood-sickness," is manifested by chronic gastro-enteritis, disturbance of appetite, fits of colic, constipation, restricted evacuation of dry dung covered with mucus or blood, fever, diarrhoea and gradually increasing emaciation and loss of strength. To these symptoms are added irritation of kidneys and bladder; haematuria, ischuria, strangury and sensitiveness in the region of the kidneys. The condition generally lasts several weeks.

*Autopsy* reveals inflammatory changes in the stomach (especially the abomasum) and in the intestinal canal. These are the most important. There are also inflammation and degeneration of the kidneys, swelling of liver and spleen, as well as signs of suffocation. The carcase is generally very much emaciated.

**Therapeutics:** After removing the cause, one must treat these cases in general as simple gastro-enteritis. Administer mucilaginous remedies, stimulate the action of the skin by friction and hydropathic bandages. To counteract increasing weakness and prostration, use stimulants. For the nephritic symptoms, tannin or sugar of lead may be given.

## POISONING BY RAPE-SEED CAKE CONTAINING OIL OF MUSTARD.

**Symptoms.**—Chronic inflammation of the bowels. Tympanites, constipation, diarrhoea, bloody faeces, cessation of milk. Signs of brain irritation : thrusting, butting, turning ; abortion.—*Autopsy* results : Haemorrhagic enteritis.—*Treatment* according to symptoms.

## POISONING BY CROTON OIL.

**Symptoms** are those of violent gastro-enteritis, with choleraic diarrhoea ; general weakness and exhaustion ; thin wiry pulse. Death occurs in one to three days.—*Autopsy* proves the presence of severe gastro-enteritis.—The *treatment* consists in mucilaginous remedies and opium.

## POISONING BY HELLEBORE.

(*Helleborus niger, viridis et fatidus.*)

**Symptoms.**—Vomiting, colic, bloody diarrhoea ; stupor, stupidity, weakness. Death occurs with spasms. *Autopsy* shows gastro-enteritis and the appearances of suffocation. *Therapeutics* : Symptomatic ; use stimulants and especially camphor.

POISONING BY WHITE HELLEBORE (*Veratrum album*) AND VERATRIN.

**Symptoms.**—The same as in preceding paragraph, only that the gastro-enteritic phenomena are weaker, and where the veratrin is outwardly applied, are altogether absent. Chief symptoms are : violent vomiting and choking, slavering ; severe pain ; continuous convulsive spasms, which are even tetanic ; great excitement, followed later by paralysis.—*Autopsy* shows all the signs of suffocation.—*Treatment* according to symptoms.

## POISONING BY ACONITE.

**Symptoms.**—Stomatitis, slavering, retching, belching, vomiting, grinding the teeth, diarrhoea and very painful colic. Inflation, weakness, paralysis, staggering, tremblings, convulsions mydriasis, loss of consciousness and tumbling. Death

### POISONING BY WATER HEMLOCK.

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in a few hours with spasms.—*Autopsy* shows gastro-enteritis and hyperæmia of the brain.—*Treatment*: According to symptoms; stimulants, especially camphor—also tannin.

### POISONING BY RANUNCULUS (*Acris, arvensis, sceleratus*).

**Symptoms.**—Choking, vomiting, slavering, colic, diarrhoea, albuminuria; staggering, stupidity, tremblings and sudden tumbling. Death often resembles apoplexy.—*Autopsy* shows gastro-enteritis and signs of suffocation.—*Treatment*: according to symptoms, tannin.

### POISONING BY THE PLANT MERCURY.

(*Mercurialis annua et perennis*, or dog's mercury.)

**Symptoms** are those of colic or gastro-enteritis; also haematuria, strangury, tenderness in the region of the kidneys; greatly accelerated breathing, rigors; small and rapid pulse. In one recorded case the symptoms of poisoning appeared ten hours after eating the herb.—*Autopsy* shows gastro-enteritis and nephritis.—*Treatment* must be guided by the symptoms (mucilaginous, anaesthetic and stimulating remedies).

### POISONING BY EUPHORBIA (Wolf's Milk, or Spurge).

**Symptoms.**—Colic, constipation, severe and even bloody diarrhoea, haematuria, tympanites, small pulse and palpitation, stupor (in cattle and sheep).—*Autopsy* shows presence of gastro-enteritis.—*Treatment* must be according to symptoms (opium and mucilaginous remedies).

### POISONING BY HEMLOCK (*Conium maculatum*).

**Symptoms.**—Slavering, foaming, endeavours to vomit; stupor, staggering, paralysis, convulsions and rapid death.—*Autopsy* reveals nothing characteristic beyond the appearances of gastro-enteritis.—*Treatment*: Tannin, stimulants, especially camphor.

### POISONING BY WATER HEMLOCK (*Cicuta virosa*).

**Symptoms.**—In addition to violent gastro-enteritic appearances, inflation, dizziness, stupidity, tumbling, actions

suggestive of hydrocephalus, weakness, prostration, paralysis ; muscular spasms resembling epilepsy, stimulated action of the heart and rapid convulsive death within 24 to 48 hours.—*Autopsy* shows gastro-enteritis, with signs of suffocation, oedema of the brain.—*Therapeutics* : Tannin, and narcotics according to symptoms, especially chloral hydrate.

#### POISONING BY FOOL'S PARSLEY (*Aethusa cynapium*).

**Symptoms.**—Slavering, retching, stupidity, stupor, paralysis, convulsions.—*Autopsy* shows gastro-enteritis.—*Treatment* : As for conium maculatum.

#### POISONING BY FLAX (*Linum usitatissimum*).

**Symptoms.**—Violent colic, inflation, diarrhoea, staggering, rigors, palpitation. Death with convulsions.—*Autopsy* reveals appearances of gastro-enteritis and the anatomical signs of asphyxia.—*Therapeutics* : Tannin, purgatives and symptomatic treatment.

#### POISONING BY NARCISSUS (*Poeticus* and *Pseudo-narcissus*).

**Symptoms.**—Intense gastro-enteritis and severe purging ; stupor and debility ; twitching and spasms.—*Autopsy* shows very severe gastro-enteritis.—*Treatment* according to symptoms.

#### POISONING BY PEPPER.

The *symptoms* are those of gastro-enteritis (in pigs).

#### POISONING BY ACORNS.

**Symptoms.**—Disturbed digestion ; in severer cases gastro-enteritis, with persistent constipation, followed by diarrhoea resembling dysentery, tenesmus, passage of blood through the anus, passage of clear coloured urine in large quantities, great lassitude.—*Autopsy* shows intense gastro-enteritis, chiefly confined to the oesophagus ; abrasion of the mucous membranes of stomach, etc.—*Treatment* according to symptoms.

#### POISONING BY ALOES.

**Symptoms.**—Colic, profuse watery diarrhoea, growing feebleness, faint, imperceptible pulse ; death in two to five days.

—*Autopsy* shows gastro-enteritis; if long continued, the inflammatory signs may be absent, so that only the emptiness and paleness of the bowels are noticeable.—*Therapeutics*: Astringent and starchily remedies.

#### POISONING BY MEZEREON, OR SPURGE LAUREL (*Daphne*).

**Symptoms.**—Stomatitis, slavering, colic, vomiting, diarrhoea, weakness; feeble, small pulse, etc.—*Autopsy* shows gastro-enteritis.—*Therapeutics* according to symptoms.

#### POISONING BY HORSE-RADISH (*Cochlearia armoracia*).

**Symptoms.**—Violent colic.—*Autopsy*: gastro-enteritis, with gelatinous infiltration of the walls of the stomach.—*Treatment* according to symptoms.

#### POISONING BY COMMON OR GREAT CELANDINE (SWALLOW-WORT). (*Asclepias vincetoxicum*.)

**Symptoms.**—Diabetes, strangury, general weakness and cachexia.—*Autopsy* shows nephritis and cystitis.

#### VARIOUS POISONOUS PLANTS.

*Belladonna*.—Mental excitement, raving, dilation of pupils, tympanites, paralysis.

*Henbane or Hyoscyamus*.—The same phenomena.

*Thorn-apple, or Datura stramonium*.—Appearances the same as in belladonna poisoning.

*Sorrel (Common)*.—Gastro-enteritic appearances, especially diarrhoea; later paralysis. *Autopsy* shows haemorrhagic gastro-enteritis.

*Rhododendron*.—Slavering, choking, vomiting, colic, bloody diarrhoea, stupor, paralysis.

*Water dropwort (*Oenanthe crocata*)*.—Stomatitis, colic, diarrhoea, general paralysis.

*Cuckoo-pint, Wake Robin, or Lords and Ladies (*Arum maculatum*)*.—Dermatitis, shivering, dyspnoea, paralysis.

*Lice-bane, or Louse-wort (seeds of the *Delphinium staphisagria*)*.—Stomatitis, colic, stupor, paralysis, anaesthesia.

*Rough Chervil (*Chaerophyllum temulum*)*.—Colic, stupor, general paralysis.

*Common yellow Melilot (*Melilotus officinalis*)*.—Paralysis of muscles.

*Male Shield Fern (*Filix mas*; extract of *filix*)*.—Vomiting, slavering, diarrhoea, loss of consciousness, uncontrollable movements, amaurosis, motor paralysis.

*Common Bracken (*Pteris aquilina*)*.—Timidity, disturbance of consciousness, tottering, paralysis.

*Cow-wheat (Molambyrum).*—Sleepiness, cerebral depression, slight colic, muscular weakness, dizziness, bloody urine.

*Poisonous Fungi—Toadstools, etc.*—Maniacal phenomena, staggering.

*Birthwort (Aristolochia clematitis).*—Gastric phenomena, colic, polyuria, staggering, somnolence, imperfect vision, muscular weakness, loss of hair. *Autopsy* reveals stomatitis, gastritis and enteritis.

*Polygonum.*—Gastro-enteritis, paralysis.

*Marsh Marigold (Caltha palustris).*—Colic, tympanites, strangury, haematuria.

*Broad-leaved Water Parsnip (Sium latifolium).*—Gastro-enteritis, excitement, stupor.

*Galega officinalis.*—A case of poisoning in sheep is reported by Blanchard.

*Potato-tops.*—Mallenders, or scab-like eczema on the legs and genitals; stomatitis, diarrhoea, conjunctivitis. Appearances sometimes resemble those of foot-and-mouth disease.

*Ferns communis.*—Bleeding at the nose, haematuria, hemorrhage from the bladder and intestines.

*Lily of the Valley (Convallaria majalis).*—Symptoms similar to those of poisoning by digitalis.

*Milk Vetch (Astragalus mollissimus).*—Brain disorders, spasms, paralysis.

*Garlic (Allium).*—Apparent frenzy, slavering.

*American Locust-tree (Robinia pseudacacia).*—Colic, tympanites, weakness, paralysis.

*Knotted Figwort (Scrophularia nodosa).*—Diarrhoea, uncertain gait, lassitude.

*Treacle-Mustard (Erysimum crepidifolium).*—Choking, restlessness, staggering, colic in geese.

*Poplar (Populus balsamifera).*—Colic, diarrhoea.

*Common Viper's Bugloss (Echium vulgare).*—Slavering, vomiting.

### III.—ANIMAL POISONS.

#### POISONING BY CANTHARIDES.

**Symptoms.**—In the digestive organs: slavering, stomatitis, difficulty of swallowing, vomiting, colic, bloody faeces and diarrhoea with tenesmus. In the urino-genital organs: frequent urination with strangury, blood, albumen and casts; excited sexual impulse and redness of the vaginal mucous membrane. General debility, staggering, convulsions, falling; small, imperceptible, wiry and accelerated pulse. Sometimes conjunctivitis.—*Autopsy* reveals stomatitis, pharyngitis, even laryngitis and acute gastro-enteritis; nephritis; cystitis.—*Therapeutics:* Symptomatic treatment of the intestinal inflammation with mucilaginous drinks, opium. Stimulants, especially camphor. *No oil.*

POISONING BY SERPENT BITE.

(*Vipera berus* and *Redii*.)

**Symptoms.**—Lassitude, prostration, restlessness, shivering, dyspnoea, abnormally rapid pulse, which at last becomes imperceptible. The course is very acute. Local swelling, which creeps gradually towards the trunk.—*Autopsy* shows dark, fluid blood and local inflammation. No other sign is constant.—*Therapeutics*: Anti-venene, cauterisation by caustics, burning of the wound. Check absorption by application of an elastic bandage. Use spirits of ammonia both externally and internally, also subcutaneous injections of permanganate of potash, bichromate of potash, chlorine water, carbolic acid or creolin. Internally alcohol and other stimulants, especially liquid ammonia.

POISONINGS BY BEE-STINGS.

**Symptoms.**—These consist of local swellings of the skin, with tendency to the formation of ulcers and necrosis, whereby sometimes considerable patches of skin become necrotic. More or less severe general phenomena are also seen, such as paralysis, stupor, septicaemia, haematuria. In cases of numerous stings in the region of the nose, suffocation may ensue as a result of the consequent swelling.—*Autopsy* reveals the symptoms of sepsis (fatty degeneration of the body muscles, clay-coloured liver, enlargement of the spleen etc.).—The *treatment* is partly surgical (antiseptics, antiphlogistics, tracheotomy), and partly internal (stimulants).

APPENDIX TO TOXIC GASTRO-ENTERITIS.

(*The Purely Narcotic Poisons.*)

**Strychnine Poisoning.**—*Symptoms*: are those of a rigid cramp (*Tetanus toxicus*) ; tetanic spasms, which last from a few seconds to several minutes, spreading over the whole body ; stiff, outstretched attitude of the extremities, neck, vertebral column and tail ; dyspnoea during the attacks ; death by suffocation. Course very acute, averaging in dogs five to seven hours, after which time the danger is over. *Autopsy* shows no essential anatomical changes ; signs of asphyxia. *Therapeutics*: The best specific is chloral hydrate (dogs two to ten grains), as well as repeated doses of chloroform carefully administered ; also morphia injections and bromide of potash may be tried. Tannin, moreover, is also to be recom-

mended as an antidote. At the outset emetics are advisable. Finally, artificial respiration may be resorted to.

**Solanine Poisoning.**—*Symptoms*: Staggering, stupor, paralysis, slower breathing; sometimes sudden fall and death in a few minutes; in other cases, the duration is from a few hours to one or two days. In hens staggering and spasms are noticed. *Autopsy* shows nothing of importance. The gastro-enteritic signs sometimes perceived do not arise from the solanine poison, but from the action upon the intestinal mucous membrane of fermenting and germinating matter.—*Therapeutics*: Tannin and stimulants.

**Prussic Acid Poisoning.**—*Symptoms*: Accelerated and laboured breathing, restlessness and fear; staggering, paralysis, stupor, shivering, twitching, spasms resembling epilepsy, vomiting, colic, diarrhoea and collapse.—*Autopsy* shows bright-red blood in acute cases, brown when chronic; smell as of bitter almonds; signs of asphyxia.—*Treatment*: Hydrated oxide of iron (see arsenical poisoning) acts as a direct antidote by formation of a non-poisonous salt; bleeding, artificial respiration, stimulants.

**Poisoning by Charcoal Fumes.**—*Symptoms*: Dizziness, stupor, loss of consciousness, asphyxia. *Autopsy* reveals bright-red blood and signs of suffocation.—*Therapeutics*: Fresh air, cold douches, blood-letting, stimulants.

**Poisoning by Darnel-grass (*Lolium temulentum*).**—*Symptoms*: In horses, chiefly cerebral; in cattle, loss of consciousness and general spasms. Sometimes appearances of colic are also noted. *Autopsy* shows slight gastro-enteritis; hyperæmia of brain and spinal cord.—*Treatment*: Tannin and stimulants.

**Alcoholic Poisoning.**—*Symptoms*: General excitement and restlessness; in cattle, even furious and maniacal signs. Increased redness of the mucous membranes of the head, palpitation, full and accelerated pulse. Later, stupor, staggering, symptoms of drunkenness, swaying about, total loss of consciousness, paralysis and collapse. Death not unfrequently results. Sometimes abortion and stimulated sexual desire. *Autopsy* shows no constant lesion. The organs smell of alcohol. Hyperæmia of the brain, and even hydrocephalus.—*Therapeutics*: Stimulants, especially coffee, ether, camphor, ammonia, carbonate of ammonia; cold douches on the head.

**Poisoning by Chickpeas (*Cicer arietinum*) and Chickling Vetch (*Lathyrus sativus, mutans*).** *Lathyrismus. Loco-disease.*—*Symptoms*: In horses roaring and difficult breathing, owing to paralysis of the laryngeal muscles; paralysis, weakness in the loins, suffocation.—*Post-mortem* reveals nothing of moment.—*Therapeutics*: Change of fodder; tracheotomy.

**Poisoning by Common Gas.**—*Symptoms*: Stupor, reeling, paralysis, convulsions.—*Autopsy* shows very light-red blood, hyperæmia of the brain. *Treatment*: Fresh air, artificial respiration, stimulants.

## *ANIMAL PARASITES IN INTESTINAL CANAL.* 211

**Poisoning by Morphine.**—Excitement, violent muscular tremblings, stupor, paralysis, weak action of the heart. *Antidote*: Atropine.

**Poisoning by Pilocarpine.**—Excessive secretion of perspiration and saliva; dyspnoea, mental excitement, paralysis.—*Antidote*: Atropine.

**Poisoning by Morphia.**—Excitement, spasms, constipation, coma.

**Poisoning by Apomorphine.**—Excitement, spasms.

**Poisoning by Santonin.**—Restlessness, cerebral depression, staggering, reddish yellow urine.

**Poisoning by Antifebrin.**—Motor paralysis, cerebral depression, somnolence, palpitation, gastric disturbances, dark-coloured urine.

## *ANIMAL PARASITES IN THE INTESTINAL CANAL (*Helminthiasis*).*

**General Remarks.**—The gastro-intestinal canal is in our domestic animals frequently the abode of bowel-worms (*entosoa*). Not all of these, however, are specially or demonstrably injurious to their hosts. The damage they cause is, first of all, dependent upon the numbers in which they congregate, and upon their species. On the other hand they often cause abnormal conditions through injury to the intestinal walls and by their strange wanderings (e.g., nematoids in the ductus choledochus). When present in great number they cause disturbance by irritation and by numerous, though small and insignificant, abrasions, as, e.g., *tænia echinococcus* in dogs; they hinder the passage of the bowel contents, and may even, by knotting themselves together, produce complete stoppage (*ascaris megalocephala* in horses). Moreover, the absorption of material for their own growth and maintenance cannot be without effect upon the animal in which they live. On the whole, then, we may consider the following symptoms caused by them as most important:

1. *Badly nourished condition (anaemia and cachexia).*
2. *Intestinal catarrh and hemorrhage of the bowels.*
3. *Stoppage, as well as symptoms resembling colic*
4. *Symptoms of brain irritation.*

There is nothing characteristic of intestinal worms in these symptoms, as they may all occur independently of them. They are, therefore, only to be attributed to worms when these are passed frequently, and, indeed, a positive diagnosis is only possible on the visible evidence of frequent passage of worms. But one must not be content even with such evidence, and

neglect a careful examination of the sick animal, lest too hasty a diagnosis be made; for there may easily be other forms of disease present which are in no way connected with the worms, and which may consequently be overlooked to the injury both of the animal and its owner. On the other hand, we must not omit to add that animals often manifest no signs during life which would lead one to suppose that they suffer from worms, and indeed appear quite healthy, even though worms be present in large numbers. The course of worm-sickness depends upon the kind and quantity of the worms in the bowels, and may have serious results, especially with young animals, owing to the condition of cachexia produced. As regards preventive measures, we are unfortunately only in a few instances as yet able to protect the animals from the invasion of worms (viz.: *taenia echinococcus*, *t. coenurus*, *t. cucumerina* and *trichina spiralis*), for in the case of most of them we cannot even suspect the presence of the eggs or larvae in food or water. Sometimes we find not only one individual, but several, and even many species of worms as causes of illness. In such cases one has to deal with a combination, so that it is impossible to lay the blame on any one individual or species. Especially in dogs and fowls we find the most diverse kinds of worms in the same animal.

**Frequency of Intestinal Worms in Dogs.**—The following statistics give exact information on this point. Out of 70,000 sick dogs received in the Berlin Canine Hospital during nine years, 1,266, or nearly 2 per cent., were treated for helminthiasis. Deffke found during his investigations in the Berlin Dog Hospital from 200 cases of dissection, that 62 per cent. of all the dogs in Berlin suffer from intestinal worms. Of these 54 per cent. showed tapeworms, and 32 per cent. round worms. The most frequent entozoa in dogs are in that city the *taenia cucumerina*. The percentage of the various kinds will be best seen from the following table:

<i>Taenia cucumerina</i> . . . . .	40 per cent.
.. <i>marginata</i> .. . . .	7 "
.. <i>serrata</i> .. . . .	5 "
.. <i>coenurus</i> .. . . .	0.5 "
.. <i>echinococcus</i> .. . . .	1.0 "
<i>Bothriocephalus latus</i> .. . . .	0.5 "
<i>Ascarides</i> .. . . .	18.5 "
<i>Nématode du rein</i> .. . . .	1 "
<i>Dochmias trigonocephalus</i> .. . . .	4.5 "
<i>Spiroptera sanguinolenta</i> .. . . .	2 "
<i>Pentastomum tanicoides</i> .. . . .	6.5 "

Schoene found by his investigations in Leipzig as follows: 1. *Taenia* (and entozoa in general) manifest themselves in the different races of dogs

in the proportions given below. Hunting dogs were in 52.94 per cent. of cases infested with worms (*T. serrata* being most numerous). Butchers' dogs to the extent of 66.66 per cent. (*T. marginata* being most prevalent). Yard dogs showed worms in 40.44 per cent. of cases (contained most *T. cucumerina*). Dogs used for purposes of draught, 72.22 per cent. (*T. marginata* most numerous). Sheep dogs were 57.14 per cent. (showed *T. marginata*, *serrata* and *cucumerina*), and were the only dogs that contained *T. coenurus* (7.14 per cent.). Of pet dogs, 70.37 per cent. were the bearers of entoscoa (having 36 per cent. of *T. marginata* and *cucumerina* and 15.7<sub>1</sub> per cent. of *T. serrata*). 2. *Ascaris marginata* were most plentifully present in pet dogs (42.1 per cent.). 3. *Strongylus trigonocephalus* were only found in pet dogs (3.2 per cent.). 4. *Echinococcus polymorphus* only in draught dogs (5.5 per cent.); *cysticercus cellulosae* and *hemistomum alatum* likewise only in draught dogs (5.5 per cent. of each). 5. *Pentastomum tenuiooides* appeared to be most plentiful in sheep dogs (as many as 21.42 per cent.).

Krabbe found *T. marginata* in 75 per cent. of all dogs in Iceland, but in only 20 per cent. in Copenhagen.

Worms are very plentiful in *Poultry*. In 18 dissected fowls Lucet found *tænia proglottina* in 18, *heterakis papillosa* in 15, *trichosoma collare* in 11, *heterakis inflexa* in 9, *tænia infundibuliformis* in 7 and *tænia cesticillus* in 6.

#### I.—TAPEWORMS.

**Varieties.**—The following kinds of tapeworm are found in the bowels of our domestic animals:—

A. IN DOGS.—1. *Tænia cucumerina*, the most frequent of all. Its larvae were proved to be in the dog-flea (*pulex sericeus*); 2. *Tænia serrata*, which springs from the cysticercus pisiformis of hares and rabbits (cysticerci in the liver, lungs and peritoneum, vulgarly called the venereal disease of hares); 3. *Tænia marginata* springs from the cysticercus tenuicollis of the ruminants and of pigs; 4. *Tænia coenurus* comes from the coenurus cerebralis of ruminants; 5. *Tænia serialis* springs from the coenurus serialis of hares and rabbits; 6. *Tænia echinococcus* springs from *Echinococcus polymorphus* of the herbivora and pigs; 7. *Bothriocephalus* species, which are rare. The larvae live in various fish (pike, perch, char, salmon-trout, grayling, sea-trout and eel-pout, or turbot).

B. IN SHEEP.—1. *Tænia expansa*, which produces the tape-worm plague in lambs, and is said to grow to 65 yards in length; 2. *Tænia ovilla*, very rare in Germany, but common in France and Italy.

C. IN HORSES.—1. *Tænia perfoliata* are most common; 2. *Tænia plicata*; 3. *Tænia mamillana*, very scarce, only about  $\frac{1}{2}$  inch long.

- D. IN CATTLE.—1. *Tænia expansa*; 2. *Tænia denticulata*;
- 3. *Tænia alba*.
- E. IN GOATS.—*Tænia expansa*.
- F. IN CATS.—1. *Tænia crassicollis*; 2. *Tænia elliptica*;
- 3. *Dibothrium decipiens*.
- G. IN RABBITS.—*Tænia leporina s. pectinata*.
- H. IN POULTRY.—1. *Tænia infundibuliformis* (hens and ducks); 2. *Tænia lanceolata* (geese and ducks); 3. *Tænia cuneata*; 4. *Tænia proglottina* (the larvæ are parasitical in *limax cinereus*, a kind of snail); 5. *Tænia cesticillus*; 6. *Tænia tetragona* (hens); 7. *Tænia cantania* (turkeys); 8. *Tænia crassula* (pigeons); 9. *Tænia fasciata*; 10. *Tænia setigera* (geese); 11. *Tænia trilineata*; 12. *Tænia coronula*; 13. *Tænia anatina* (ducks); 14. *Tænia gracilis*; 15. *Tænia sinuosa*; 16. *Tænia megalops*; 17. *Tænia conica*; 18. *Tænia imbutiformis* (ducks); 19. *Tænia tenuirostris* (geese and ducks); 20. *Bothriocephalus longicollis* (fowls).

**Natural History.**—Tapeworms (cestodes) belong to the group of flat worms and form colonies (strobiles), whose individual members develop by budding from the scolex, which is the "nurse," or "head" of the entire colony. The riper members, which are capable of reproduction, are called proglottides. The head possesses a retractile trunk (rostellum) and attaches itself by suckers and hooks (armed tapeworms) to the intestinal mucous membrane. Each proglottis consists of an external muscular rind with an outer skin (cuticula), and an inner, which contain the generative organs and the vascular trunk. The sexual organs are hermaphrodite and consist of uterus, ovary and yolk-sack, of seminal vessels and vagina on one hand, and of testicles, spermatic duct and cirrus on the other. The sexual opening is usually lateral on the free margin. The eggs, which are found in the uterus in extraordinary quantities, have a very hard, membranous covering in several layers, and contain a globular, bright embryo, furnished with four or six hooks; they are very resistant, but may be destroyed by drying. If these eggs in any way reach the stomach of another animal, an embryo with four or six hooks is developed which perforates the wall of the stomach and wanders into various organs, where, after losing its hooks, it becomes enclosed in a bag (encysted) and changes to a "bladder-worm" (cysticercus). In this "bladder-worm" the embryo has already become a scolex and the progenitor of a

later colony. Should it reach the stomach of a congenital animal, the scolex is set free by the digestion of the bladder, fastens itself to the side of the stomach and develops into a colony of tapeworms.

The mode of development just described, in which the bladder-worm only represents the larval stage, that is to say, the sexless forerunner of the tapeworm in its various habitats, has been experimentally demonstrated by van Beneden, Küchenmeister, von Siebold, Leuckart, Haubner and others.

**History of the Development of *Tenia Cucumerina*.**—This is very interesting. Melnikow maintained formerly that the larva of this *tenia* was contained in the dog-louse (*trichodectes canis*). Contrary to this we have already shown that the origin of this, the commonest of all tapeworms, must be another, seeing that these lice are but seldom found in dogs. But Grassi has lately discovered that the dog-flea is really the host which transfers the *tenia cucumerina*. The larvae of tapeworms are also found in the human flea (*pulex irritans*). The embryos exist free inside the body of the flea, often as many as 50 in one flea. The dog is infected by swallowing the flea. Young dogs whose mothers harbour fleas show tapeworms five to ten days after birth. As fleas often change their residence by jumping, dogs may also be infected which are themselves free from fleas. The true preventive, then, against this tapeworm is the destruction of the fleas.

***Tenia Echinococcus*.**—The investigations of Müller and Mangold appear to confirm the earlier supposition of Ostertag, that there are two kinds of *T. echinococcus*, corresponding to the two kinds of *echinococcus* (*cysticus* and *multilocularis*). The common *tenia* belonging to the *E. cysticus* sort has stout hooks, the rarer or *multilocularis* shows slenderer hooks and a globular accumulation of eggs.

***Tenia Serialis*.**—Railliet has shown that, in addition to *T. coenurus*, another tapeworm occurs in dogs, viz.: *T. serialis*. Its relative, the *coenurus serialis*, shows an arrangement of heads in a row, and grows as large as a hen's egg, inhabiting the connective tissue of the muscles, the subcutaneous and subserosa (peritoneum) of wild and tame rabbits, hares, squirrels and beavers.

**Pathological Changes produced by Tapeworms upon the Intestinal Mucous Membrane.**—The presence of these worms in the bowels, especially when they only occur singly, often produces no catarrhal change on the mucous membrane, or only a very slight one. But in other cases very intense catarrhal and inflammatory effects are caused. Schieferdecker found on examining dogs infested by *T. cucumerina*, singular tunnels in the mucous membrane of the small intestine, through which the linked chains of *tenia*

travelled like railway trains. These tunnels were formed by the interlacing and growing together of the intestinal villi above the tænia. The villi were many of them prolonged to four or five times their usual length, covered over with epithelial matter which had peeled off, and were hypertrophied within by a considerable new growth of capillary vessels. The "Lieberkühn" follicles were mostly much shortened near these hollow ways and pressed asunder in their upper parts. Sometimes one also found an increase of the inter-tubular connective tissue in the form of distinct septa. Such changes must cause more or less severe functional trouble, not only by disturbing resorption by the hypertrophic villi, but also by checking intestinal secretion through atrophy of the glands. The most serious pathological changes of the intestinal mucous membrane were wrought in dogs by *tænia echinococcus*. These little worms, which have only three or four segments, often hook themselves by their sharp hooks on to the membrane in hundreds of thousands, producing intense and frequently fatal inflammation with haemorrhage of the bowels. Other tapeworms may by accumulation in masses completely stop up the lumen of the bowels and cause intussusception. In *Poultry*, the changes wrought upon the intestinal mucous membrane consist in loosening and hyperæmia, as well as in a desquamative catarrh, with profuse secretion of a yellowish-red, purulent mucus. Sometimes also tubercle-like nodules are found in the intestinal wall (*nodulæ tædiasis*, noticed by Moore and Morton).—Mégnin has noticed perforation along with formation of diverticles and cysts in the small intestine of horses produced by *Tænia perfoliata*. Cadéac found the duodenum of a dog perforated by two specimens of *T. serrata*. A similar case of bowel-perforation was noticed by Lahogue. In each case signs resembling rabies were noticed during life. Wolpert found specimens of *T. serrata* in the oesophagus and kidneys.

Tapeworms in dogs are generally found in the small intestine. Of the *T. cucumerina*, which is commonest in dogs, 15-25 individuals will inhabit the jejunum and ileum (the maximum is over 100, but in one case Neumann found 767 !); but generally there are not more than one or two. *T. echinococcus* occurs in dogs in hundreds of thousands, so that one square centimetre of mucous membrane contains often 100 or more. Ascarides are sometimes found singly in dogs, and sometimes in greater numbers (25 to 250).

**Symptoms.**—In the majority of cases no particular

signs betray the presence of tapeworms in the bowels. But if illness be produced, it generally takes the form of chronic intestinal catarrh, to which other symptoms may be added according to the kind of dog. In practice, tapeworm disease is most important in dogs, lambs and poultry.

These worms are very often present in dogs, without any external sign of ailment. Butchers', shepherds' and hunting dogs are most exposed to their ravages, and young dogs are decidedly more subject to their invasion than older ones. When the tapeworms reveal their presence to the eye, one mostly finds the symptoms described under chronic catarrh of the bowels. Sometimes these are accompanied by a ravenous hunger, without the animal gaining anything in weight, indeed rather the contrary. The appetite is very changeable, young dogs not seldom manifest great restlessness, whine, run hither and thither, snap towards their hind quarters, rub themselves along on the haunches, frequently change position, etc. On inspecting the anus we often find in it, or on the neighbouring hairs, fresh or dried proglottides, sometimes in large numbers. In very excitable dogs, or when the tapeworms occur in large quantities (especially *T. echinococcus* and *T. cucumerina*), sometimes very severe and violent pain may supervene, which fact formerly led to the opinion that genuine rabies could be produced by the presence of tapeworms, and even to-day may now and then cause one to suspect rabies. The symptoms of this "pseudo-lyssa" are dizziness, twitching, epileptic fits and paralysis, as well as sundry appearances very much like rabies; inclination to bite, change of voice, paralysis of the lower jaw, great lassitude and prostration. Leisering produced the same symptoms experimentally by feeding a dog with *echinococcus* cysts. Similar signs have been noticed after feeding with *T. cucumerina*. Their explanation lies in the very painful and usually fatal inflammation which they cause in the bowels. The accumulation of tapeworms in the intestines may sometimes cause constipation, and may even result in death. Perforation of the intestinal walls is also occasionally seen. Leslie reports on a very interesting case of self-infection in a dog by *T. serrata* (*cysticercus pisiformis* in the brain). The symptoms were grinding of the teeth, frantic running about, etc.

2. In *Sheep*, tapeworm sickness shows itself as the so-called tapeworm plague among lambs, especially in wet summers, and on damp, marshy meadows. The predisposing cause

for reception of the *tænia* lies here in the youthfulness of the animals. In older sheep a previous gastric weakness seems to produce this pre-disposition. The *tænia expansa* is remarkable for its rapid growth. According to Spinola, lambs of four weeks may contain tapeworms 12 yards long, which has led to the unfounded supposition of an ante-natal infection. The phenomena of tapeworm plague have often nothing characteristic, and are not to be distinguished from those of cachexia, viz., chlorosis, pale skin and conjunctiva, and light-coloured wool, which can easily be plucked out, and is poor in fatty matter. Later on, serious disturbance of digestion follows; appetite and rumination both irregular; the young animals are retarded in their growth, appear dull and feeble, may easily be caught, linger behind the flock and lose flesh rapidly. The constipation which usually results causes slight signs of restlessness; they keep lying down and jumping up again, arch their backs, and make vain attempts to evacuate, hold up their tails, run suddenly away and are not infrequently more or less tympanitic. Should the cachexia become far advanced, diarrhoeic discharges ensue, with great prostration. In this condition the lambs are quite incurable and finally perish of exhaustion.

3. In *Poultry*, with whom tapeworms are most numerous, the trouble shows itself partly in gastric disturbances, especially increasing diarrhoea, with slimy and even blood-stained faeces, and partly in steadily persistent emaciation and slowness of growth, although the appetite is well maintained. The creatures are thereby sad and languid, have lustreless and ruffled plumage, which usually harbours a quantity of parasites (mites, etc.), have at times fits resembling epilepsy, and often stand for a long time as though asleep, and then, seeming to awaken, run about and feed. But gradually they cease to eat, grow very feeble, fall down and die very quickly. The sickness is at times almost epidemic. Friedberger describes an extensive outbreak among pheasants. Lucet noticed an enzootic appearance of *tænia setigera* among geese. The symptoms were emaciation, fetid diarrhoea, paralysis and rapid death, the mortality being 20 to 30 per cent. (In one goose, 600 individuals of the *tænia setigera* were counted.) A similar outbreak among young geese caused by *T. lanceolata* has been described by Ellinger (convulsions, spasms as of epilepsy, extreme diarrhoea and mostly sudden death).

Tapeworms in the other domestic animals (horses, cattle,

goats, cats, rabbits) more rarely give rise to illness. Where they do, the appearances are essentially as above described. Mégnin and Waldmann report on cases of perforative peritonitis in horses caused by *T. perfoliata*. Hurlimann found immense quantities of the same tænia in a horse suffering from paralysis. Hendricks describes a case of enzoötic intestinal inflammation produced in foals by *T. plicata*. Haubold and Truelsen mention two cases of *T. denticulata* in cattle; the symptoms were emaciation and chronic tympanites. In cats an epidemic of tapeworm plague occurred in 1874 in the Black Forest (*T. crassicollis*), during which the field mice also disappeared (*cysticercus fasciolaris*), probably from the same disease. The ailing cats grew rapidly thin and finally perished. In several cats which died of gastritis, Zschokke found *T. crassicollis* in the stomach, and attributed death to their presence there, as in healthy cats which he examined they were always in the small intestine.

**Diagnosis.**—This can only be positively made when proof is shown that tapeworms have been passed, and in sheep frequently only by *post-mortem* examination. The phenomena of chronic intestinal catarrh give no certain guidance. Tapeworms are usually evacuated through the anus along with the faeces, and are then found on the surface of the dung-balls singly or numerous, and often movable sections, which, in dogs, creep about near the anus, stick to the hairs and finally dry up to thin yellowish or whitish flat objects. More rarely larger or smaller sections of tapeworm are vomited. In consequence of the irritation produced about the anus by the proglottides dogs frequently rub along on their hind quarters to relieve the itching. But in other complaints they do the same. The dilation of the pupils, which has by many been considered a sign of helminthiasis, has no diagnostic importance.

**Differential Diagnosis of the various kinds of Tapeworm.**—On this point and for their classification we must refer our readers to the text-books of general pathology and of parasitology. We will here merely point out the chief microscopic differences of the species most commonly found in dogs.

1. *Tænia cucumerina*: Frequently appear knotted together, the mature proglottides looking like pumpkin seeds, i.e., elongated and elliptical in shape, reddish, with two sexual openings on each free margin, at most  $\frac{1}{8}$ th of an inch wide, 2 to 4 inches long (rarely 8 inches), and moderately thick. Under the microscope reddish egg-heaps are seen (so-called cocoons).

2. *Taenia serrata* : The proglottides united to each other like saws (hence the jagged edges) ; one sexual opening placed alternately left and right on the free margin ; members very long (up to  $\frac{1}{2}$  an inch) and broad (to  $\frac{1}{4}$  of an inch) ; barrel-shaped ; total length from  $1\frac{1}{2}$  to 3 feet.

3. *Taenia marginata* : Proglottides joined together in squares, often broader than long, with straight edges and alternating lateral sexual openings. Total length, 2 to 5 yards. These are the broadest, longest, and also stoutest tapeworms of the dog.

4. *Taenia coenurus* : Proglottides quadrangular, the hindermost oblong, and very long in proportion to width. Colour, white. Total length,  $\frac{1}{2}$  to 1 yard.

5. *Taenia echinococcus* : Only has three links (seldom four) ; about  $\frac{1}{2}$  of an inch long ; only the last link or member contains eggs ; links are white, elliptical, and may be mistaken for intestinal villi.

These various proglottides may be microscopically recognised by the form of the uterus, and their heads by the differences in shape of their knobs.

**Therapeutics.**—There is a great variety of remedies in use for tapeworms, but all of them have to be administered in relatively large doses, and if they do not themselves possess purgative properties, no time must be lost between loosening the adherent heads and driving out the entire colony, as otherwise the scolices will re-attach themselves ; an evacuant must therefore be given either along with or soon after the anthelmintic. Treatment should be preceded by preparatory measures. These consist in fasting the animal for a day and emptying the bowels either by enemas or purging medicines, in order that the remedies may come into different contact with the worms. All worm remedies, then, should be given on an empty stomach. In the case of dogs it is well to have a second dose handy, as the first is so often vomited.

1. *For Tapeworm in Dogs* use extract of male fern, 10 to 90 grains in capsules or pills, in the latter case generally with male fern powder, which may also be given alone (freshly powdered) in doses of from 2 to 6 drams. The extract of male fern is one of the most reliable taeniafuges. Then Kamala also has a good effect ( $\frac{1}{2}$  to 4 drams), combining purgative with anthelmintic properties. Areca nut (2 $\frac{1}{2}$  to 5 drams) grated in pills or mixed with fresh butter ; kousso (2 to 6 drams) stirred up in milk and repeated in an hour ; pomegranate root bark (1 to 12 drams) boiled in water. Finally pumpkin seeds are also recommended (25 to 30 seeds), oxide of copper (1 grain), chloroform, creolin and other specifics.

2. *In the Tapeworm plague among Lambs* it is first of all

necessary to avoid all dangerous or suspicious pastures. All the remedies suggested for dogs may also be used here in appropriately modified doses. Of newer specifics, picrate of potash (7 to 20 grains) in pills, and also kussin ( $1\frac{1}{2}$  grains) have been tried with good results. Others prefer for sheep the extract of male fern in doses of  $\frac{1}{2}$  to 1 dram, or kamala (1 to  $1\frac{1}{2}$  drams). According to Trasbot the simplest and least injurious remedy is feeding with pine-tree sprouts (which contain oil of turpentine), twigs or berries of the juniper, all of which the young lambs eat greedily. At one time Spinola's worm-cakes were given, they contain tansy, wormwood and tar, 1 part of each, salt  $\frac{1}{2}$  part, made into a cake with flour and water; also Chabert's oil (*oleum contra teniam*, a distillation of animal oil and spirits of turpentine), which was given in tea-spoon doses along with 3 to 5 grains of tartar emetic.

3. *For Tapeworms in Poultry* Gurn recommends powdered areca nut as the best remedy (30 to 45 grains made into pills with butter) but turkeys are said to be somewhat excited by it. Purgatives are seldom needed. Powdered tansy root (in doses of 15 to 45 grains) is said to be much inferior to areca nut. Mégnin recommends a mixture of ginger, gentian, fennel, aniseed, coriander and aloes (aa a pinch for each bird).

All the above remedies may be used for the other domestic animals if required. For *horses*, the medicines mentioned in the next paragraph on the Ascarides are to be preferred, viz., tartar emetic, arsenic and spirits of turpentine, and areca nut in addition (two dessert spoonfuls with each feed).

## II.—ROUND WORMS OR ASCARIDES.

**Natural History Notes.**—Among ascarides in our domestic animals we must specially mention the following :—

- (a) In *Horses and Asses* : *Ascaris megalcephala*.
- (b) In *Dogs* : *Ascaris marginata* and *ascaris mystax*.
- (c) In *Cats* : *Ascaris mystax*.
- (d) In *Cattle and Pigs* : *Ascaris lumbricoides*.
- (e) In *Poultry* : *Heterakis* (formerly *ascaris*) *inflexa*, and *heterakis vesicularis*, *dispharagus nasutus* in hens, *heterakis maculosa* in pigeons, *heterakis dispar* in geese.

The history of the development of ascarides is not yet

clear. Formerly the opinion was held, based upon negative attempts at direct transmission, that they pass through the body of some intermediate host. But Grassi has lately proved, by experiments in feeding, that the *ascaris marginata* is produced direct, without any intermediary, by ingesting the eggs from the faeces of animals afflicted with worms. This is confirmed by the observation also made by Grassi, that young dogs which were carefully isolated with their mother (already infected with round worms) were found to be infested with *ascaris marginata*, their only nourishment having been their mother's milk. We also have seen these worms in dogs a few weeks old, and Penberthy reports the same (a dog two weeks old was infested with ascarides; in another of six weeks 250 were counted).

**Pathological Changes Produced in the Intestinal Mucous Membrane by Round Worms.**—These worms, which are found sometimes in thousands, inflict severer injury upon the internal membranes than tapeworms, their heads being armed with toothed mouths. Indeed, they occasionally perforate the intestinal walls and produce peritonitis. Dogs may die quite suddenly from ascarides, and on *post-mortem*, one finds on the inflamed and swollen membrane small, round, black-red points, which mark the places occupied by the worms, and show raised, wall-like edges with ulcerous indentations. In other cases signs of intense haemorrhagic enteritis are found; occasionally the ascarides have formed passages and channels with ulcerous, eroded, and thickened walls in the mucous membrane. This process may extend through all the coats of the intestine. Often, too, the worms knot themselves together in balls and completely block the lumen of the bowel. Several cases of perforation by *ascaris megalcephala* in horses have been described in which, at the point of attachment of the mesentery, small openings with thick, indurated margins were located, the wall having at this point less power of resistance. These openings led to bladder-like cavities filled with pus, dissolved food and ascarides between the two thickened layers of the mesentery. On perforation of these intra-mesenteric abscesses, an ichorous peritonitis was developed. Several observers have found a single round worm in the swollen and thickened orifice of the ductus pancreaticus. Ortman found seven in the distended biliary ducts of a pig. In poultry, the intestinal mucous membrane is more

or less catarrhally inflamed, ulcerated and covered with thick, purulent mucus.

**Symptoms.**—Those caused by round worms differ only slightly from the signs of tapeworms. In general, they are disturbed digestion and nutrition, constipation, diarrhoea, tympanites, etc.; emaciation and certain signs of nervous irritation—itching, rubbing, facial contortions, etc.

1. In *Horses* these worms often occur without visible symptoms. In other cases they produce only slight gastric trouble, such as diarrhoea and constipation, accompanied by intermittent colic and gradual wasting. And yet worms may cause death by obstruction or perforation of the bowels. Occasionally they produce quite remarkable signs of illness. Thus, Freminet noticed a pronounced tetanus-like condition in a mare (trismus, see-saw movements, prolapse of the nictitating membrane), produced by reflex action, and which vanished on the use of worm remedies. Truelsen saw epileptiform spasms in a mare, which also disappeared as soon as the worms were driven out. Damitz found, in one case, paralytic weakness of the hind legs, produced by a peculiar affection of the spinal cord, and this, too, quickly passed away after administration of worm remedies.

2. In *Dogs* the symptoms are about the same as with tape-worms. But sudden death not infrequently occurs, both in dogs and cats, from bowel-perforation. Poison has in such cases usually been suspected, and the true cause only discovered on *post-mortem*.

3. Among *Poultry*, pigeons are especially liable to epidemics of disease caused by *heterakis maculosa*. The presence of worms reveals itself by diarrhoea, increased thirst, weakness, paralytic signs, apathy, wasting, atrophy of the pectoral muscles, drooping wings, ruffled plumage, loss of feathers, etc. Sudden fatal ending is not uncommon. Sometimes as many as 500 *heterakis* are found in the intestines of one pigeon. *Heterakis inflexa* produces the same results in hens. Sometimes it finds its way into their eggs, by wandering into the fallopian tubes. In turkeys inflammation of the coecum has been noticed (Rätz).

In other animals, ascarides are of less clinical interest. Descamps reports a case of *ascaris lumbricoides* in a sucking calf, which manifested during life much tympany and peculiar

movements of the jaws, and whose entrails contained 3½ gallons of ascarides.

**Therapeutics.**—The best remedies for ascarides, and for round worms in general, are :—

1. *For Horses* : Tartar emetic, 2 to 4 drams, dissolved in drinking-water, or with bitter remedies as an electuary, to be given three or four times, at intervals of three hours ; Santonin, for full-grown animals 2 to 4 drams, administered along with calomel ; Areca nut 3 to 8 ounces, a table-spoonful grated over each feed ; Arsenic in the form of pills, 15 to 30 grains, with bitter purgatives ; oil of turpentine, 3 to 6 ounces per dose, in form of emulsion, foetid animal oil (3 to 8 drams), benzine (1 to 3 ounces), male shield fern, tansy, absinthe, gentian, asafoetida and creolin. As an easily obtainable domestic remedy, we recommend beetroot and carrots, as well as raw potatoes (up to 2 gallons a day with wheat bran).

*For Dogs* : Santonin—grown dogs 1 to 3 grains, younger ones ½ to ¼ grain, with sugar, and followed by castor oil, or in solution. Areca nut may also be used (2 to 5 drams), extract of male fern, picrate of potash, creolin, etc. Domestic remedies : decoction of garlic in milk, per os or per anum.

3. *For Poultry*, Zürn recommends areca nut, viz. : for hens 45 grains, and pigeons 15 grains. The same remedy also for pigs (1 to 4 drams per diem).

### III.—STRONGYLES (*Palisade-worms*).

The following occur in the intestines of our domestic animals :—

- (a) In *Horses* : Strongylus armatus and tetracanthus.
- (b) In *Sheep* (and *goats*) : Strongylus contortus (tapeworm plague), strongylus hypostomus and cernuum, strongylus filicollis, strongylus venulosus.
- (c) In *Cattle* : Strongylus radiatus, inflatus, ventricosus and convolutus.
- (d) In *Dogs* : Strongylus (dochmius) trigocephalus.
- (e) In *Cats* : Dochmius balsami felis, dochmius tubeformis.
- (f) In *Pigs* : Strongylus dentatus (*sclerostomum dentatum*), strongylus rubidus.
- (g) In *Rabbits* : Strongylus strigosus.
- (h) In *Poultry* : Strongylus tenuis and nodularis (*geese*), strongylus pergracilis (*pigeons*).

I.—*STRONGYLUS ARMATUS AND TETRACANTHUS IN HORSES.*

**Natural History.**—1. *S. armatus* appears under two forms in horses: (a) as unripe, sexless larvae in aneurysms of the anterior mesenteric artery (see Embolic colic); and (b) as mature strongylus armatus in the cæcum and colon. The eggs of the worm, after being passed out with the dung, develop into free nematodes (*rhabditids*) in water and mud. Should these reach the horse's bowels, through its drinking water, they seek to penetrate to the blood-vessels, especially to the trunk of the anterior mesenteric artery, which lies nearest. Hence they roam, after several changes of skin, back to the bowels in fully matured sexual condition (to the cæcum and colon), where, by means of their trephine-like armature of the head, they fix themselves.—2 *S. tetracanthus* becomes encysted in the submucosa of the bowels and wanders thence, fully matured, into the intestinal lumen.

**Pathological Changes in the Bowels.**—1. *S. armatus* causes, wherever it is fixed (by suction), bluish-red spots on the membrane, which may often be mistaken for ecchymoses, but under the microscope reveal a reddish worm, about  $\frac{1}{16}$  to  $\frac{1}{8}$  inch long, as their centre; a slight enteritis is also produced. 2. *S. tetracanthus*, when numerous, produces, particularly in foals, haemorrhagic enteritis with minute submucous nodules (cysts), in the middle of which are yellow pus foci, with strongylus larvae. Small scars are also often left on the mucous membrane. The worms are frequently found in masses in the intestines. Cobbold found sometimes more than 150 cysts per square inch of surface in the colon.

**Symptoms.**—Illness caused by mature worms is rare in horses, and it seems that it requires a large number to produce sickness. They most frequently excite a condition of the mucous membrane of the large intestine which resembles dysentery, with consequent colic, which may even result in death. Violent straining and bloody, liquid excreta are noticed, and the arm is smeared on insertion into the rectum with a quantity of minute strongyli. Death then sometimes follows from haemorrhage of the bowels. Chronic cases occur occasionally, marked by much wasting and anaemia with severe diarrhoea. According to Schwarzmaier, diarrhoea is rife in wet summers among suckling foals, when death occurs from enormous immigrations of

*strongylus armatus*. In England of late years, many fatal cases have arisen from strongyli, both in blood-horses and cart horses (Walley, Penberthy). Heill reports cases in which meningitis and hydrocephalus internus with consecutive symptoms of immobility, were produced by worms penetrating to the surface of the cerebral cortex. Cansy speaks of atrophy and sclerosis of both testicles caused by invasion of those organs. Thrombosis of the left coronary artery, with sudden death, was observed by Cadiot in an ass; and haemorrhage of the lungs in a horse by Michalik. Kitt found sexually mature strongyli in the peritoneum of two horses; Duncan the same in the abdominal muscles, and Pütz in the testicles of a foal.

**Treatment.**—Owing to difficulty of diagnosis this is only rarely possible. In general it is the same as for ascarides. Penberthy found oil of turpentine effective. According to Spooner, palisade worms (strongyli) are difficult to drive out. Probably he alluded to the encysted larvae of *S. tetracanthus*.

Millach found from his investigations into the development of *Sclerostoma armatum* that they were either females or hermaphrodites—no males being discovered. From the ova of these sexually mature individuals there apparently sprang a fresh generation, which was both male and female. He denies that their development depended on passage through the blood-vessels, and considers it improbable that the rhabditides could penetrate, as such, into the blood-vessels, their mouths not being armed, but that the small females and hermaphrodites could do so. He also demonstrated the existence of an intermediate generation of *Sclerostoma tetracanthum*.

Cobbold has thrown much light on the development of *S. tetracanthus*. The young worms, on quitting their cysts, form cocoons of mucous matter exuded from their skin, these may be found plentifully in the faeces of their hosts (as many as 40). The embryo forms in damp, warm media a few days after shedding this skin. The larva, which resemble rhabditides at this time, may live for many weeks in the open. But an intermediate host is needed for their further development. The faeces are ingested with green fodder or at pasture. They bore into the mucous membrane of the caecum and colon, to become encapsulated there after a second shedding of the skin. In time they break through their cysts and reach the lumen of the bowels as mature worms of both sexes. There they shed their skin for a third and last time and form the above-named cocoons. They attain their definite form and sexual maturity in the colon of their host.

## II.—STRONGYLOSIS IN SHEEP (*Strongylus contortus*).

**Etiology.**—This worm (*S. contortus*) occurs in vast numbers in the abomasum of sheep, especially of lambs and

yearlings (but also in goats), and produces an affection which has been called the red plague of stomach-worms (red verminous gastritis). In economic importance it is not far behind the tapeworm plague. It occurs in spring and summer, and spreads like an epidemic, and arises especially in the neighbourhood of standing water, usually occurring along with husk or hoose (*strongylus filaria*). From this fact, Gerlach concluded that they had a joint origin and sought to prove the statement. It is doubtful if strongylosis can occur after exclusively dry feeding.

**Post-mortem Notes.**—Dissection shows the mucous membrane of the abomasum thickly covered with strongyli, whose intestines are red with the blood they have absorbed. The membrane is also in a state of chronic catarrh, and at times is perforated like a sieve. It must be noted that, owing to their being very quickly digested in the carcase, it is not easy to prove their presence some time after death. On the other hand, an autopsy is often necessary for the formation of a certain diagnosis.

**Symptoms.**—These cannot be differentiated from those of tapeworm plague. They consist in anaemia, chlorosis, pernicious anaemia with poikilocytosis, gastric attacks (especially diarrhoea), along with an ever-increasing emaciation, weakness and cachexia.

**Therapeutics.**—Use oil of turpentine, foetid animal oil or Chaberts' oils, picrate of potash (15 to 45 grains), etc.

### III.—DOCHMIASIS OF DOGS (*Dochmias trigonocephalus*).

**Etiology.**—This occurs especially among hunting-dogs which live in packs, and has been described by French and Italian veterinary surgeons under many names: dochmiasis, uncinariosis, ankylostomiasis, pernicious anaemia, bleeding of the nose, etc. It is caused by *dochmiasis trigonocephalus* (*Uncinaria trigonocephalus*), along with which, *dochmiasis stenocephalus* is usually present. Besides these, *trichocephalus depressiusculus* is said also to co-operate. A similar parasitic anaemia has been observed in men (miners and workers in tunnels);

**Autopsy.**—The parasites are mostly found in the cæcum

and small intestine. In slight cases, numerous dot-like haemorrhages are found on the mucous membrane. In the middle or to one side of the small coagula several small, thread-like worms may be seen,  $\frac{1}{2}$  to  $\frac{3}{4}$  inch long, white, and with a black longitudinal line. In severer cases, the membrane is much thickened, and penetrated by fairly large blood foci. In prolonged cases, the dochmii are found singly, even in the colon, while their number in the small intestine has decreased. The carcase also shows signs of anaemia and cachexia.

**Symptoms.**—The disease develops gradually with signs of growing weakness and wasting. In spite of a comparatively good appetite, the animals are spiritless and dull, go back in condition, develop a rough, scaly coat and a mucous, purulent nasal discharge. Later on, the characteristic signs of dochmiasis appear, viz.: bleeding at the nose and oedematous swelling of the limbs. The former is usually intermittent with shorter or longer intervals, and the amount of blood in each discharge reaches three ounces or more. Towards the end, follow loss of appetite, persistent diarrhoea, severe anaemia, inflammation of the lungs, haemorrhages, ulcers and even gangrene of the skin, accompanied by great weakness. The poor brutes are wasted to a skeleton and die at last with the signs of coma, after an illness lasting from a few months to a year. The disease may continue for years in a pack, and most of the hounds suffer from it, one after the other. It is distinguished from ordinary anaemia by the enzootic presence of dochmiasis, and by the *post-mortem* discovery of parasites; also by the success attending the use of worm-remedies. Contrary to the nose-bleedings produced by pentastoma, dochmiasis is usually accompanied by grave general symptoms, particularly that of severe anaemia. It is distinguishable from leucæmia by absence of swelling in the lymph glands, by microscopical examination of the blood; and from distemper by discovery of the oval ova of dochmiasis in the faeces under the microscope.

**Therapeutics.**—The first points to observe are—isolation of the affected animals, removal and destruction of faeces containing the parasites, cleaning the kennels and all food utensils, keeping the beds dry and giving none but pure (if possible boiled) water. Infection occurs oftenest through licking up dirty water containing ova or larvæ of the dochmii.

Medicinal treatment consists in the administration of anthelmintics. Extract of male fern, kamala, arsenic, etc., are recommended. Mégnin gives a mixture of kamala 1 dram, calomel 7 grains and arsenic  $\frac{1}{2}$  grain. Careful dieting, especially feeding with meat, must accompany the above.

Grassi and Generali have described a similar affection in *cats*, *pigs* and *poultry*, manifesting itself by extreme diarrhoea, uncontrollable vomiting, wasting, weakness, anaemia, etc.—In *sheep*, *strongylus hypostomus* produces similar disturbances, with colic. The small intestine was found quite covered with small dot-like haemorrhages, and the intestinal contents were chocolate colour. Bass noted over 50 fatal cases in an outbreak among sheep (diarrhoea, wasting, etc.).

A new strongyle was discovered by Ostertag in the abomasum of cattle (*S. convolutus*). It occurs in 90 per cent. of all cattle. Grey spots, the size of a lentil, occur on the swollen mucous membrane, which is red and flecked. These are found under the microscope to be strongyli rolled together. He says that, if present in undue numbers, they cause cachectic dropsy, which is revealed after slaughter in the form of "oedema of the cellular tissue," or "a watery condition of the flesh." They seem to make their appearance in October and November.

Hassall and Stiles give the name of *Strongylus rubidus* to a new kind of worm which they found in Washington in the stomachs of 25 to 75 per cent. of all pigs examined. Sometimes the thick mucus of the stomach was stained blood-red by these worms alone.

#### IV.—*OXYURIDES (Bodkin-Tails, Whip-Worms).*

**Species.**—The following occur in our domestic animals:—

- (a) In *Horses*: *Oxyuris curvula*, *vivipara*, and *mastigodes*.
- (b) In *Dogs*: *Oxyuris vermicularis*.
- (c) In *Rabbits*: *Oxyuris ambigua*.

**Symptoms.**—These worms are more annoying than injurious. On quitting the cæcum and colon they adhere to the rectum, causing proctitis, accompanied by itching, rubbing, and even scabs upon the tail. The diagnosis depends on finding the creatures on the faeces or adhering to the hand when inserted, or on identifying their ova in the scab-like crusts which form about the tail. Sometimes single worms may be found hanging out of the anus. Another species, the *oxyuris mastigodes*, has been found in horses after eating *leontodon taraxacum*. This sort, which is not very rare, differs from *oxyuris curvula* in its larger size, and the remarkable length of its very thin tail (females measure up to 5½ inches, of which 4½ are tail); also in its very sticky eggs.

The Treatment for oxyurides consists in clysters of soap-suds,

vinegar, weak solutions of sublimate (1-1½ per 1,000). Internal administration of anthelmintics is never needed.

#### V.—GIANT ECHINORRYNCHUS (*Echinorrhynchus gigas*) IN PIGS.

**Natural History Notes.**—According to Schneider's investigations, the immature larvae of the giant worm are ingested by pigs, in the larvae of bots or canker-worms, or perhaps through May-bugs or cock-chafers themselves, into whose bodies they have penetrated. In America, according to Stiles, the larvae of lachnosterna are the intermediate hosts.

**Autopsy.**—The pathological changes which these worms produce in the small intestines of pigs are very considerable. With their mouths armed with thorny hooks, they bore deeply into the mucous membrane, causing much inflammation, and even perforation of the bowel, with ensuing peritonitis. The small intestine feels as though filled with a bead necklace, and yellow nodules of pus, the size of hemp-seed coated with red, shine through. These on being opened are found to be foci of inflammation, with the head of the parasite for centre, and surrounded by a shell of thickened, highly reddened mucous membrane. Sometimes they have matured into ulcers, reaching often as far as the serosa. In other places the membrane is slate-grey in colour, thick and covered with mucus.

**Symptoms.**—These consist in loss of appetite, constipation, great restlessness, rubbing on the ground, burrowing, snapping at the hind quarters, increasing emaciation, twitchings, convulsions and spasms, resembling epilepsy. In young pigs, death occurs sometimes in three to four days, with the last-named symptoms. Not unfrequently the disease runs through a whole herd.

**Therapeutics.**—The first thing to do is to destroy the May-bugs, or to avoid the pastures they haunt. Internally the various anthelmintics may be used. Kocourek obtained very good results from oil of turpentine (teaspoonful doses), and the use of the following aperients: sulphate of magnesia 2½ drams and aloes 1 dram.

#### VI.—THE LARVAE OF GASTROPHILUS IN HORSES (Bots).

**Natural History Notes.**—Of the larvae of the stomach-fly (*gastrus*, *gastrophilus*), four different species occur in horses,

viz. :—1. *Gastrus equi*, or the common horse-fly, with its seat in the cardiac half of the stomach; 2. *Gastrus pecorum*, or cattle-fly, which lives as a parasite in the stomach and duodenum; 3. *Gastrus haemorrhoidalis*, or rectum-fly, which infests the same parts as No. 2; and 4. *Gastrus nasalis*, the nasal-fly, dwelling chiefly in the duodenum. These large flies, which swarm in the meadows during summer, lay their eggs between June and September on hairs of horses. From these there crawl small maggots in three to five days, which are mostly licked off by the horse, and thus reach its stomach, where they bore into the mucous membrane with their armed heads, and remain there until they issue as perfect larvæ (after about nine months). In spring they pass out with the excreta, change to their chrysalis form in the faeces, and, after three changes of skin, emerge in four to seven weeks as sexually ripe, winged insects.

**Symptoms.**—These larvæ are found regularly in the stomachs of pasturing horses, where they are generally quite harmless. Only in isolated cases and under peculiar circumstances can they cause any harm.

1. If present in large numbers, they may, in rare cases, produce disturbance of digestion and signs of colic, by exciting a traumatic gastritis. Such a case has been described by Kersten. In a foal, which had suffered from colic and gastric disorder, the stomach was found on dissection to be almost full of *gastrus* larvæ, and the membranes much inflamed.

2. In isolated cases, perforation of the stomach-wall, with resultant fatal peritonitis, or adhesive peritonitis, has been observed. Reports are also published of large abscesses between the coats of the stomach, and fatal cases of haemorrhage of the stomach.

3. The larvæ wander at times in various directions. Their most dangerous incursion is into the brain, causing inflammation of that organ, rotatory movement, cerebro-spinal meningitis and attacks resembling apoplexy. Others wander occasionally into the larynx (dyspnoea and choking); into the rectum (colic and prolapse of the rectum); into the bladder (colic, retention of urine); into the subcutis, etc. The adhesion of these larvæ to the pharynx and pharyngeal cavity cannot be designated a pathological condition, because they are often as harmless there as in the stomach.

In view of their harmlessness, treatment is generally needless,

especially as the usual worm-remedies have no effect upon them. Perroncito and Bosso recommend, as the result of experiment, carbon bisulphide, in doses of 5 drams, as the best remedy for horses. When the larvæ invade the brain, treatment is generally impossible.

**Larvae of Cestrus in Dogs.**—The larvae of *gastrophilus equi* have occasionally been found in the intestinal canal of flesh-eaters, especially in the stomachs of dogs, where they hook themselves fast and flourish. French also noted an *cestrus* larva in the scrotum of a dog.

#### VII.—VARIOUS OTHER INTESTINAL PARASITES.

**A. TREMATODES:**—*Amphistomum conicum* in the first stomach of cattle, buffaloes, sheep and goats. Usually innocuous, except in Australia, where it appears to work great devastation among the herds and flocks.

*Hemistomum alatum*, in the small intestine of dogs.

*Gastrodiscus polymastos*, in the large intestine of horses, mules and asses, in Egypt and the Island of Guadaloupe.

*Distomum echinatum* (ducks, geese), *oxycephalum*, *ovatum* (hens, geese), *lineare*, *dilatatum*, *pellucidum*, *armatum*, *communitatum* (hens), *cuneatum* (peacock).

*Monostomum mutabile* (geese), *attenuatum* (geese), *verrucosum* (hens, geese, ducks).

The trematodes produce in poultry similar gastric disturbances to round and tapeworms. Treatment is also the same.

**B. SPIROPTERA:**—Among these we may notice:—*Spiroptera megastoma* and *microstoma*, in the stomachs of horses. The former cause nodules in the cardiac portion of the stomach, ranging in size from a bean to a walnut, and each having an opening. These may cause gastritis, colic, etc.

*Spiroptera sanguinolenta*, in the stomach and pharynx of dogs, in knotted clumps. May also produce gastritis. According to Grassi, the black beetle (*blatta orientalis*) is an important intermediate host of this worm. These beetles being very common in Southern Italy, the spiroptera also frequently infest the dogs there. Raillet states that the worms sometimes penetrate to the bronchial glands, to the lungs, into the walls of the aorta and even to the neighbourhood of the kidneys. In one case the bronchial lymph-glands were found greatly hypertrophied and infiltrated with purulent centres, containing spiroptera. The symptoms noticed during life were: difficult swallowing, vomiting, diarrhoea and extreme emaciation. In another case, a dog showed symptoms like those of rabies, and

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autopsy revealed worm-clusters in stomach, bowels and bronchial tubes, with inflammation of the duodenum.

*Spiroptera strongylina*, in the stomach of pigs.

*Spiroptera scutata*, under the pharyngeal mucous membrane of cattle, sheep, goats and pigs (identical with *myzomimus scutatus* Müller, and *gongylonema pulchrum* Molin.)

C. *GNATHOSTOMA HISPIDUM* the "hand-shield" stomach-worm of pigs. This worm produces verminous gastritis in pigs. Csokor found the serosa of the base of the stomach greatly infiltrated and covered with dark spots as large as poppy-seeds, the mucous membranes thickened to two inches and covered with small nodules. In it were forty worms, whose bristly heads reached as far as the serosa, where they caused the above-named spots. The same condition has been noticed in Hungarian swine.

D. *TRICHOSOMA TENUISSIMUM* occurs in incredible quantities in pigeons (1,830 have been counted in one bird). It causes severe intestinal catarrh, chronic and bloody inflammation of the bowels, wasting anaemia, etc.

E. *TRICHOSOMA CONTORTUM* causes indigestion of the crop in ducks.

F. *TRICHOCEPHALUS AFFINIS* in cattle, sheep and goats; *trichocephalus depressiusculus*, in dogs; *trichocephalus crenatus* in pigs. All these are harmless.

G. *FILARIA UNINCATA*, the cause of "filariasis" in ducks, occurs also in geese and swans. According to Hamann, the intermediate host is the water-fly (*daphnia pulex*). The worms lodge in clusters,  $\frac{1}{4}$  inch in diameter, in and beneath the mucous membrane of the stomach. Zürn states that they may excite violent inflammation of the oesophagus, stomach and intestines. Klee found filariae under the mucous membrane of the lower end of the oesophagus and of the stomach in swans, where they formed tumours the size of cherries.

H. *ŒSOPHAGOSTOMA COLUMBIANUM* causes the so-called nodular disease of the stomach in sheep and cattle (Japan and Arkansas). Dinwiddie is the authority for the latter statement. The larvae, which are  $\frac{1}{5}$  inch in length, live in the intestinal wall, where they form nodules, in size from a millet grain to a pea. The mature worms, about  $\frac{1}{4}$  inch long, live free in the bowels.

I. *DISTOMUM HETEROPHYES* is rather common in Egypt, where it causes obstinate diarrhoea. Janson says that in Japan it occurs in dogs.

## CHAPTER IV.

### DISEASES OF THE LIVER.

#### JAUNDICE. ICTERUS.

**General Notes.**—Jaundice like colic is not a disease of itself. They are both merely symptoms of diseases. Authors have described :—1. A *catarrhal* jaundice, caused by catarrh of the biliary ducts; 2. A *hepatogenous* form, which sometimes appears as an accompanying symptom of liver complaints (parenchymatous, interstitial hepatitis, atrophy of the liver, congested and amyloid liver, fatty liver tumour-formations—such as carcinoma and sarcoma of the liver—distomatosis, echinococcus or hydatid sickness, thrombosis of the portal vein, etc.). To this latter form catarrhal icterus strictly belongs. 3. In contrast to these two forms of jaundice, which from their manner of development are also called congestive, resorption, retention, or mechanical icterus, and which are always referred to a disordered liver, there is yet a third form in which the yellow discoloration is traced to decomposition of blood in the process of septic, infectious or toxic diseases (sepsis, murrain, influenza, anthrax, poison, etc.). The existence of a true *haematojenous* jaundice has, however, been disputed. Of these three only catarrhal icterus can be treated as a separate disease, as in the two latter cases the icteric symptoms are something secondary, and may even be absent altogether.

**Etiology of Catarrhal Icterus.**—This complaint is by far most frequent in dogs, and rarely occurs in horses. Among 70,000 sick dogs treated in the Berlin Dog Hospital in nine years 102 suffered from icterus (0.15 per cent.). It arises from the swelling and consequent constriction of the mouth of the excretory duct, produced in the course of duodenal or gastro-duodenal catarrh, whereby the biliary duct and liver are surcharged with

bile, and the bile is reabsorbed by the liver and passed into the blood. The causes, therefore, of catarrhal icterus are the same as those of gastro-duodenal catarrh, and consist mostly in errors of diet, the eating of damaged food; the influence of irritants (mechanical, thermal, chemical, or infectious) upon the mucous membrane of the small intestine; in congestion of blood caused by heart weakness; and in sympathetic affection of the intestinal canal during feverish infectious diseases.

**Autopsy.**—Along with the catarrhal changes in stomach and bowels, one finds the mouth of the biliary duct opening in the intestine stopped up or constricted by the swelling of the duodenal mucous membrane. The membrane of the other part of the biliary duct is, on the other hand, only rarely changed. The biliary passages are found much enlarged (we have seen one in a dog which was dilated to the thickness of a finger); the gall bladder is very full and distended, and even the smallest, most microscopic gall capillaries are distinctly injected. The bile, at the same time, often thickens to a crumbling, brittle mass. In the excretory bile-duct a firm mucous plug is sometimes fixed. The liver is enlarged, infiltrated with bile, and anaemic; the liver-cells are charged with pigment, and, if the disease last long, become atrophied (biliary cirrhosis). The bile can only be with difficulty removed by pressing the bladder.

All organs, except the peripheral nerves, the white matter of the brain, the spinal cord, and the tissue of the cornea, are more or less deeply coloured yellow. The carcase is anaemic. In the blood the white blood-corpuscles are more numerous, while the red vary greatly in size. Fatty degeneration of the myocardium is invariably present; the kidneys are anaemic; the epithelium of the kidneys in the extended tubuli uriniferi and loops has undergone fatty degeneration and is permeated by yellow-brownish granular pigment. Finally, haemorrhage is found in various organs, especially in their mucous membranes.

**Symptoms.**—The appearances of catarrhal icterus follow on those of gastro-duodenal catarrh. They are, therefore, generally preceded by disturbance of appetite, vomiting, coated tongue, increased thirst, diarrhoea, fever, etc. Then jaundice is developed, at first slight, but growing steadily more severe. The sclerotic conjunctiva and eyelids first show the characteristic yellow colour, varying shades from deep citron to

orange. Later, the other visible membranes take on the colour, especially that of the mouth, and the skin (in sheep and dogs), also the urine, sweat, milk, etc. Specially characteristic is the dark yellow, or citron, or brownish-yellow, or greenish-yellow colour of the urine, which stains white blotting paper dipped into it and may be extracted by shaking up with chloroform. It is due to the presence in the urine of biliary pigment in greater or less quantity. The chemical proof of its presence is best established by Gmelin's method with fuming nitric acid. This is slowly added in diluted form to the urine, when a change takes place wherever the two fluids are in contact from green to blue, violet and red. Of other methods, Fleischl's may be named, in which the urine is mixed with a concentrated solution of Chili nitrate of soda, and then with concentrated sulphuric acid. This also produces the same colour-changes. According to Rosin, a 10 per cent. alcoholic solution of tincture of iodine is sprinkled on the urine, on which a grass-green ring is formed round each drop as it falls. Besides colouring matter one often finds albumen, though generally only in small quantities, also grains of pigment, drops of fat, diffusely-stained epithelia of the urinary duct, short urinary casts deeply infiltrated with pigment, and also biliary acids. In our chapter on gastro-duodenal catarrh we have fully discussed the fact that in dogs biliary pigments appear in the urine as a consequence of intestinal catarrh, yet without producing any discolouration of the mucous membranes; but in horses the reverse is the case, viz., jaundice of the membranes without the presence of biliary pigment in the urine.

While the absorption of these pigments into the blood leads to jaundice, that of the biliary acids in severer cases may lead to much more serious symptoms. First it produces retardation of the pulse and fall of bodily temperature by several degrees (to 97°, 93°, and even 90° F.). Moreover, by reaction upon the nervous centres, lassitude and weakness are produced, dulness of the sensorium, and in horses frequently distinct symptoms of immobility. A further series of troubles is caused by the absence of bile in the bowels. For instance, constipation sets in, because the action of the bile on the peristalsis is lacking, and the quantity of fluid in the bowels is lessened by the same cause. The faeces often stink quite unbearably, their colour is lighter, and in dogs which are fed on meat are grey or clay-coloured, because the fatty matter is not extracted by the bile as usual from the contents of the bowels, and urobilin is consequently

absent from the faeces. Enlargement of the liver only rarely occurs in domestic animals.

The *duration* of catarrhal icterus is very different, according to the kind of animal affected. In dogs, owing to the total stoppage of the excretory bile-duct, the end is very often fatal. In horses, on the contrary, the prognosis is distinctly more favourable. Generally the disease runs several weeks. In the more severe cases with dogs (*icterus gravis, cholæmia*) haemorrhagic diathesis sometimes ensues, with haemorrhage in the skin, on the mucous membranes and in the retina.

According to Latschenberger, it is rarely possible to prove the presence of biliary pigment in fresh horse-urine by Gmelin's process, even when actually present. It causes a dark-brown zone which hides the characteristic colour-change. He therefore first dilutes the urine freely with water, adds a solution of baryta and lets all stand twelve to twenty-four hours; removes the muddy fluid above the precipitate and applies Gmelin's test to the latter. The colour reaction is, however, in this case only weak.

**Therapeutics.**—The treatment of catarrhal icterus corresponds with that of gastro-duodenal catarrh, and consists in strict adherence to a rational diet (flesh meat for dogs, green fodder, carrots, etc., for horses), and in the administration of anti-catarrhal salts, the best of which is artificial Carlsbad salt. Also aloes, castor oil, and tincture of rhubarb are recommended. Drastic remedies should be avoided, as they increase the swelling. Attempts have been made to reduce this swelling by using tannin with red wine as an astringent, but with what success is uncertain. Mechanical manipulation is certainly more effectual. This includes (with dogs) emptying the gall bladder by pressure from the abdominal wall, and cold water injections into the rectum. Siedamgrotzky succeeded in faradising the liver transversely through the abdominal walls. Finally, Rancilla recommends lead balls to remove the invagination, which is frequently present. To combat the weakness, somnolence and coma which occur during the latter days, and may end in death, stimulants must be used (camphor, coffee, ether).

The so-called "Icterus of the New-born" (*icterus neonatorum*), often noticed in human beings, appears not to be rare among domestic animals. Hartmann saw foals in Babolna in which jaundice was either present at birth, or after five or six days, and traces these cases generally to inflammation of the umbilical veins, with liver disease. In any case temporary constipation (no-delivery of the meconium) combined with weak blood-pressure may cause icterus in the newly born. The prognosis, according

to Hartmann, is unfavourable, as most of those who suffer from it die in a few days from exhausting diarrhoea. He recommends alkalis with rhubarb as a remedy. In the slaughter-houses, calves are often found suffering from jaundice.

So-called *Icterus gravis* (*cholomia*) in dogs is, in many cases, caused by complete stoppage of the excretory bile-duct by a mucous plug, or equally often by invagination of the duodenum; less frequently, however, by pressure of the enlarged spleen, or by neoplasm of the pancreas. Here poison seems also often to co-operate, especially moulds, or phosphorus and common salt (bad meat, herring-brine). In other instances, acute bacterial atrophy of the liver seems, as in men, to be the origin. The course of cholomia is extraordinarily rapid, death following with rapid sinking of strength and of bodily temperature. The jaundice is very marked, the urine becomes dark citron-yellow, and the skin is also yellow, particularly on the lower parts of the body. Haemorrhage may set in later on the mucous membranes.

#### RUPTURE OF THE LIVER (LIVER-APOPLEXY).

**Etiology.**—A rending of the liver with shattering of the hepatic tissue and haemorrhage into the abdominal cavity may result from strong mechanical force. It occurs after a violent fall or wrench, e.g., during colic, after contusion in the region of the liver, after excessive work or over-exertion, such as leaping. But, as a rule, such mechanical force is not alone sufficient to cause rupture of the liver; that organ must first be predisposed by certain changes of the parenchyma and vessels, or particularly by amyloid degeneration of the liver. (See paragraph thereupon.) Stress has lately been laid upon the connection between this last and rupture of the liver, on the ground that amyloid degeneration produces a lessened power of resistance and greater friability of the vessels. In just the same way fatty degeneration of the liver induces rupture, as do also hyperemic and inflammatory affections. Over-feeding may in lambs predispose to rupture by fattening the liver. Lastly, haemorrhage of the liver occurs after embolism of the hepatic artery and obstruction of the hepatic veins in the form of so-called haemorrhagic infarcts; further, during the course of infectious diseases and poisoning (anthrax and murrain, or poisoning by phosphorus) all of which result in fattiness of the liver-cells, or in parenchymatous hepatitis, and also in connection with certain new growths in the liver, e.g., with cavernous tumours, melanomata and cancer.

**Autopsy.**—The post mortem conditions in haemorrhage of the liver are somewhat simple. When only superficial, the tissue

of the liver is found to be softened and changed to a tender, blackish-red substance. The serosa of the liver is sometimes raised in the form of blisters above the peripheral blood-focus. In horses rupture occurs in the serosa, through which the blood gushes into the abdominal cavity, mostly on the convex side, and on the lower edge of the liver. The margins of the rupture are found covered with coagulated blood. When the haemorrhage is more deeply seated the parenchyma of the liver is frequently infiltrated with numerous blood foci, which consist of thick blood and crushed liver tissue. In haemorrhagic infarcts wedge-shaped, sharply-defined infiltrations are produced. The above-named degenerative changes may also be found in the liver. Liver-abscesses may also be sequelæ of haemorrhage of the liver.

**Symptoms.**—In rupture of the liver these occur either suddenly, without premonitory sign, or in connection with certain pre-disposing liver affections, such as icterus, constipation, pain on pressing the region of the liver, etc. The aspect is precisely that of internal haemorrhage :—The animals show great anaemia of the mucous membranes, restlessness, sweatings, cold extremities, weak and finally imperceptible pulse, with palpitation, staggering, falling, dilated pupils, rolling of the eyes, sudden blindness, amaurosis, etc. In severe cases of bleeding, death often follows very quickly—in a few minutes. Slighter haemorrhage may be resorbed, or lead to fatal peritonitis. In one case, observed by Benjamin, of a horse kicked by another in the region of the liver, polyuria occurred in three days and lasted for two months, i.e., until death. On dissection the left lobe of the liver was found to be thickened, tender and infiltrated with an abscess:

**Therapeutics.**—When the course is rapid no treatment avails. In protracted cases absolute quiet, combined with the use of astringents : Ergot, sugar of lead, etc., may at least be tried. If weakness increase, as a result of loss of blood, one may also try stimulants (camphor, ether, alcohol, etc.).

## PARENCHYMATOUS HEPATITIS.

**Etiology.**—Parenchymatous hepatitis, i.e., inflammation of the secretory liver-cells, occurs in all domestic animals. Its causes are partly the same as those quoted for congestive

hyperæmia of the liver, viz., damaged fodder, mechanical lesions, excessive heat, infectious disease, or poison. Clinically the disease is of subordinate importance, partly owing to its difficult diagnosis, and partly because it usually represents secondary changes in various infectious diseases or poisoning.

**Autopsy.**—The anatomical changes consist in enlarged liver, a bluntness of the edges, very soft and friable consistence, pale yellow, clayey colour, perihepatitis, adhesion of the lobes of the liver to each other, or of the liver to the diaphragm, enlargement and granular projection of the several acini above the surface of the rupture, opaque swelling, granular and fatty degeneration of the liver-cells, with pigment infiltration, hyperæmia of the interacinous connective tissue, as well as in formation of foci of softening, and of numerous small (or single large) haemorrhagic foci in the parenchyma of the liver, or under the serosa, and which may produce rupture of the liver.

**Symptoms.**—These are very irregular. Along with jaundice and the signs of feverish indigestion, pains have been observed in the passage of faeces, caused probably by compression of the liver by the diaphragm and abdomen, as may be proved in smaller animals by pressure in the region of the liver.

**Therapeutics.**—Treatment consists in the use of antiphlogistic remedies, such as neutral salts, and in regulation of the diet.

**Acute Yellow Atrophy of the Liver.**—This is a very acute and pernicious form of parenchymatous hepatitis, and is most frequently observed in lupinosis, with which, in most of the cases described, it is probably identical. The so-called "liver-typhus," in sheep and horses, described by Haubner, Franzen and others, is also mostly an acute yellow atrophy of the liver, caused by mashed food, or by grazing in inundated pastures. Parenchymatous hepatitis also changes into acute atrophy after septicæmia, phosphorus poisoning, or after several infectious diseases. Autopsy reveals diminution (atrophy), softening and yellow discolouration of the parenchyma; the liver-cells have fallen into a fatty, granular degeneration. Extreme icterus of the liver (acute "yellow" atrophy), or, in many places, also considerable hyperæmia (acute "red" atrophy) may likewise be found. In the other organs various hemorrhages appear. The symptoms correspond entirely with those of lupinosis (gastric disturbances, icterus, cerebral symptoms). Thus, in horses, Adam noted at first, poor appetite, slight jaundice, reddish-brown urine; and later, signs of depression, staggering, tremblings, slower pulse; and finally, intense icterus, with signs of immobility, accelerated pulse—and all this within from 25 to 48 hours.

## PARENCHYMATOUS HEPATITIS.

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**Suppurative Hepatitis (Liver-abcess).** This is also a form of parenchymatous hepatitis, which rarely occurs among our domestic animals. The abcesses may have various origins :—1. Infection may come from the bowels, or metastasis from the arteries of the liver, e.g., in pyämia, glanders and other acute infectious diseases ; 2. Thrombosis of branches of the portal vein or the umbilical vein (omphalophlebitis, so-called "navel-ill" in foals and calves) ; 3. Intrusion of foreign substances (fodder, barley-ears, sand, parasites), coming from the stomach, bowels or blood ; 4. Mechanical injuries, inflammation proceeding from the bowels, gall-stones, etc. Hepatic abcesses are sometimes spread over the parenchyma of the liver in considerable numbers (during formation of metastasis), at other times singly, and only rarely are they confluent. Most of the pus foci are surrounded by a zone of thicker, dark-brown liver-tissue. Sometimes fully developed haemorrhagic infarcts can be perceived, which are wedge-shaped, with central softness. In cattle we sometimes find echinococcus cysts which have discharged their contents. The symptoms of metastatic liver abcesses are chiefly rigors, irregular and persistent fever, icterus and digestive disorder. At times they discharge their pus into the abdominal cavity.

**Necrosis of the Liver.**—Peculiar tumour-like nodules with necrotic centres have in late years been found in cattle, pigs and sheep, which have been termed necrosis nodosa multiplex hepatitis, and are traceable to intrusion of the bacillus of necrosis into the parenchyma of the liver. Nodular foci are found on the liver, which organ is much enlarged. These foci are limited in area, vary in size from a pea to a walnut, are round, hard and are found on section to be dry, spotted, grey-yellowish and often surrounded by capsules of hard connective tissue. Their appearance is often accompanied by perihepatitis and peritonitis fibrinosa. Berndt, who observed 15 cases, all ending in death, says that the symptoms in cattle are clearly marked. The animals show diminished appetite, sluggish, stiff movement, undoubted enlargement of the liver, much sensitiveness in the region of that organ and of the entire abdomen, high fever ( $104\text{--}106^{\circ}$  F.), constipation, strained breathing, inability to stand, and finally they die in three to five days, after a prior appearance of liquid, tar-like diarrhoea, accompanied by sinking of the internal temperature. Berndt only noted the complaint in cows which were advanced in pregnancy or had recently calved.

**Hypæmia of the Liver.**—The complaint is difficult to diagnose in our domestic animals, and its causes are manifold. But one must not confuse congestive hypæmia with that arising from engorgement. The former is normal in every digestive process, and occurs in abnormal manner in many disorders of the digestion, e.g., after over-eating, or too little exercise (fat dogs), or after taking spoilt, irritant or mouldy food. Moreover, it occurs at the beginning of a liver-inflammation, after violent shock, excessive exertion, intense heat, or in warm, moist and swampy districts and steamy stables. But passive hypæmia (caused by engorgement) is partly traceable to heart-failure (valvular defects, especially of the mitral) ; partly to chronic lung-troubles (emphysema of the lungs, interstitial chronic changes of the lungs, compression of the same by hydro-thorax, etc.). Finally, hypæmia of the liver occurs as a secondary

symptom in infectious diseases, such as anthrax, inflammation of the adjoining intestinal canal, cachectic conditions, or intrusion of parasites into the liver. Autopsy shows the hyperemic liver to be at first swollen and enlarged, darker in colour, roughly granulated (when broken) and relatively firm in consistence. The centre of its lobules is dark, their periphery light. If the disease last long, a so-called nutmeg liver results, in which, through the alternation of lighter and darker parts, it acquires the appearance of nutmeg. This arises from enlargement of the surcharged central hepatic veins, with consecutive compression, fatty degeneration, pigmentation and finally atrophy of the liver-cells. During the course of the chronic form, haemorrhage may occur in the parenchyma of the liver with singular pigmentation of the same.

The *phenomena of hyperæmia of the liver* are remarkably uncertain, and it is only rarely identified as a disease, but generally confounded with others, and especially with gastro-intestinal catarrh. Of more important symptoms (which only too often are absent), we might name sensitiveness of the liver-region in dogs and a slight icterus, caused by compression of the bile-duct by the dilated hepatic veins when the liver is congested. But it is often impossible to decide whether it be a case of hyperæmia of the liver, or a hepatitis already developed. In intense hyperæmia, rupture and haemorrhage of the liver may both occur. Treatment should consist in regulation of the diet, place of abode and exercise, as well as in the use of mild, opening salts, among which artificial Carlsbad salt deserves the first place.

**Inseptic Hepatitis in Young Pigs.**—Semmer describes an outbreak near Dorpat of hepatitis in pigs, the origin of which was traced to a certain micrococcus. Infection is said to occur after birth through the navel. The liver-changes produced are: enlargement, nodular surface, hyperæmia alternating with anaemia; and, on section, the appearances of "nutmeg liver," enlargement, fatty degeneration and pigmentation of the liver cells, with cellular infiltration round about them. The disease can be transmitted by inoculation to rabbits, guinea-pigs and white rats. Period of incubation, 20 to 47 days. Symptoms: Loss of appetite and apathy, which only appear a short time before death.

#### CHRONIC INTERSTITIAL HEPATITIS (*Cirrhosis of the Liver*).

(*Chronic Induration of the Liver.*)

**Etiology.**—Chronic interstitial hepatitis appears as a slow inflammation of the connective tissue of the liver, with shrinkage and atrophy of the true secretory liver-cells. It occurs most frequently in horses as the so-called Schweinsberger sickness, but also in dogs, cattle and pigs. Its causes are not yet known. With horses, the blame is laid on swampy country and irritant plants, whose poison is supposed to act upon the liver like alcohol upon that of man. In dogs, cirrhosis of the liver appears to be connected with valvular defects of the heart (engorged liver).

**Post-mortem.**—According to the stage reached by the disease, this reveals either a diminished and shrunken liver (atrophic cirrhosis), or one enlarged by new growths of connective tissue (hypertrophic cirrhosis). The consistence is always harder and firmer, for, by the added connective tissue, the liver often becomes leathery or cartilaginous and creaks under the knife. In protracted cases, the surface is mostly granular, nodular and even lobulated; the serosa opaque and thickened. At first, the colour is that of a nutmeg, and later very light-yellow, and the entire organ is bloodless. On cutting it, a white, large-meshed net of newly-formed connective tissue is seen, more or less broadly streaked, and which includes larger or smaller areas of fatty-degenerated and atrophied parenchyma of the liver, and separates the hepatic lobes by callosities of varying widths. Microscopically investigated, the process consists, at first, of infiltration of small cells in the periportal connective tissue and subsequent intra and extra-vascular development of similar tissue therein. Besides these changes in the liver we find hyperæmic engorgement and chronic catarrh of stomach and bowels, enlarged spleen, ascites, anasarca, the cerebral membranes fully charged with blood, haemorrhage in various organs, and furthermore, in Schweinsberger sickness, excessive dilation of the stomach.

**Symptoms.**—These unfold very slowly. The ailment generally begins imperceptibly with chronic digestive disorder, in which constipation alternates with diarrhoea, and the animal grows visibly thinner. Then follows slight jaundice, tenderness of the liver when pressed, and (in dogs) ascites and anasarca. With horses, a peculiar dulness of the sensorium is superadded, with staggering and other signs of immobility, and also periodic fits of colic. The causes of these cerebral disturbances are obscure. They may lie either in re-absorption of the bile-producing elements of the blood (acholia), or in a complication with chronic pachymeningitis. At first there is no fever, but later on feverish symptoms arise—low to medium ( $103^{\circ}$  F.). The duration is from a few months to several years. The disease is not unfrequently mistaken for immobility.

**Therapeutics.**—There is no cure known, but palliative treatment consists in the use of alkalis and neutral salts, especially Caribad salt, aloes and diuretics. Suitable diet, green fodder, etc., must accompany these.

**Schweinsberger Sickness.**—This ailment of horses, which reminds one in many points of lupinosis, is a quite peculiar form of chronic interstitial hepatitis. It was first noted at Schweinsberg, in the valley of the Ohm, in the Electorate of Hesse, and later, in many other parts of Germany. It is everywhere bound to certain conditions of soil and of the plants growing thereon, being strictly local and enzootic in swampy, peaty districts which are subject to frequent inundation. Probably it arises from the gradual action of a certain irritant poison, first upon the liver and later on the brain (after the analogy of alcohol, phosphorus or lupine-poison). But the causes are not exactly known. In the Glon valley, clover-feeding is spoken of as the cause; but farms lying on higher ground escape, and horses of middle age (eight to ten years) are chiefly attacked. But later accounts state that hill-side farms and horses of all ages are attacked, and in Schweinsberg the sickness often carries off every horse in a stable within one year. The signs are at first unnoticeable, or mistaken for chronic gastro-intestinal catarrh. The animals show diminished or debased appetite, lick and gnaw the woodwork, yawn, are dull and depressed and have fits of colic. After weeks and months, jaundice sets in (but not always), the pulse is quickened, and peculiar attacks resembling immobility or staggers occur, to which are added periodical colic, constipation, complete abstinence from food, with medium fever. Emaciation keeps pace with the progress of the disease. Temporary signs of improvement are seen sometimes, especially after a change of pasture; but the symptoms return with increased severity, and the strength fails rapidly, with oedematous swelling of the extremities. Death follows in several weeks, or up to nine months, but only very rarely occurs in three or four days. Occasionally rupture of the stomach brings a more sudden ending.

Autopsy reveals the changes of the liver already described. These are principally an inflammatory hyperplasia of the interstitial connective tissue, especially that lying between the lobes, with localised fatty degeneration of the liver-cells. Sometimes the liver is double its normal size. Great distention of the stomach is also often found, as well as the aspect of chronic gastro-intestinal catarrh (slate-coloured discolouration and hypertrophy of the mucous membrane). Treatment is, as a rule, hopeless. Ganter recommends disinfection of the stable as a prophylactic. The intra-tracheal injection of Lugol's solution has been found useful by several practitioners. As the animals, during the early stages of the disease, move about freely and show few or no signs of illness when at work, they are generally quickly sold by their owners, who know only too well what to expect in the future.

#### AMYLOID LIVER.

**Occurrence.**—Amyloid degeneration of the liver has so far been found in horses, dogs, cattle, sheep, rabbits and poultry, either alone, or in conjunction with amyloid kidneys, spleen or bowels.

**Etiology.**—Its causes must be sought mostly in cachectic

conditions, chronic inflammation, and permanent ulcerative processes. Rabe found in half the cases of amyloid liver in horses the simultaneous presence of chronic pleuritis, pericarditis, or peritonitis. He also observed it along with hydrothorax and diffuse persistent formation of abscesses in the muscles. Caparini found amyloid degeneration of the liver in horses, side by side with exudative pleuritis and increasing cachexia, combined with orchitis and phlebitis. Bruckmüller also identified the disease in cattle, in which it was combined with chronic nephritis. Anomalies in feeding appear furthermore to be connected with its origin. Thus Bruckmüller noticed the appearance of amyloid liver among horses fed on malt and kept in very restricted quarters. It is clear, therefore, from the above, that the causes of amyloid degeneration of the liver are identical both in animals and man.

**Autopsy.**—The liver is generally enlarged and has thick and rounded edges. Its consistence in horses, contrary to what is found in man, is soft, doughy, pulpy, or crumbling. Moreover, the otherwise smooth serosa shows in cases of longer duration an over-lying rind. The liver is greyish-brown in colour, and contains but little blood. The amyloid centres are sometimes evenly spread, and at others irregularly. In hens they also occur as disseminated nodules. Under the microscope the liver-capillaries, and especially the intra-acinous vessels, appear thickened, of homogeneous and glistening consistency, and crumbling away into flakes. This amyloid degeneration can also be demonstrated chemically. The parts just named take on a mahogany-red stain when treated with Lugol's solution of iodine, shading from grey to violet blue in dilute sulphuric acid and to aniline red in methyl violet. The true liver-cells only share partially in the amyloid degeneration; but most of them, on the other hand, are atrophied and infiltrated with fat. In addition to the liver, amyloid degeneration shows itself in the spleen, kidneys and bowels.

**Symptoms.**—These are of very uncertain character. In general a probable diagnosis can be made, if cachectic and anaemic conditions, combined with tumour of the liver and albuminuria (amyloid kidneys), be found. Icterus occurs only in isolated cases. The disease generally ends fatally in liver-rupture, with symptoms of internal haemorrhage into the abdominal cavity.

**Therapeutics.**—Treatment is quite hopeless and can only be directed against the primary disease.

#### CANCER OF THE LIVER.

**Pathological Notes.**—This occurs most frequently in old and fat dogs, more rarely in horses or other domestic animals. It appears partly as a metastatic and partly as a primary cancer. The liver is then more or less enlarged, with uneven surface and interspersed with nodules, ranging in size from a millet-seed to a man's fist. Sometimes these are isolated, at others confluent, or again are arranged in rows, and have usually a soft consistence, a pale yellow colour, and are often fairly rich in blood, with a soft or calcareous centre. Cancer nodules are then usually found in the portal system of the liver, as well as in the liver itself; also in the spleen, kidneys, pancreas, omentum, lungs, etc. Sarcomata of the liver occur also.

**Symptoms.**—These are very uncertain and obscure. Frequently they consist only in signs of gastro-intestinal catarrh or slight icterus. A probable diagnosis is only possible in dogs and cats by palpation of the liver-region, which sometimes reveals an irregular, lumpy surface of that organ, as, for instance, if an external growth of carcinoma be present (e.g., on the mamma). Among consecutive symptoms of liver-cancer in dogs, is the development of ascites; and in horses the appearance of colic pains caused by peritonitis, as well as the addition of rupture of the cancerous nodules, which are frequently infiltrated with large blood-centres. This generally causes death by bleeding into the abdominal cavity. Extreme emaciation shows itself as the disease advances, even to muscular atrophy, which, in dogs, is most marked about the head.

#### GALL-STONES.

**Etiology.**—The occurrence of gall-stones and their consequent morbid conditions (cholelithiasis) are, among animals, clinical rarities, although rather frequent among men. The explanation of this difference does not lie in any lesser predisposition on the part of the former to the formation of stones. On the contrary, experience shows that urinary calculi are just as frequently found in domestic animals as in man, and in dogs perhaps more so. There appear then to be other reasons.

From a dietetic standpoint we must remember that in man an irregular or abnormal manner of life and lack of due exercise occasion manifold disturbances in the region of the mesenteric vein, and may also be a predisposing cause of cholelithiasis. Such causes do not operate in our domestic animals, above all, not in those that work. The fact that gall-stones are especially rare in horses is explicable on the ground of their regular feeding and constant exercise. The absence of the gall-bladder in the genus *equus* may also have something to do with their comparative immunity; and its presence has undoubtedly a contrary effect in other animals (men, dogs, cats and cattle), for in this reservoir engorgement and consequent decomposition of bile may easily occur. Any accumulation of the bile results first in a thickening of the fluid and later in precipitation of its component salts, or of other matter, which in normal and healthy bile should be held in solution (cholestearin, bilirubin).

Gall-stones occur in domestic animals, both in the gall-bladder and in the gall-ducts, and are most common in cattle and dogs. The smaller ones vary from the size of poppy-seed to that of a pea, are round, oval or roller-shaped, and sometimes worn on their surfaces. Birnbaum counted 400 such small stones in a horse. The larger ones may attain 4 inches in length, by  $1\frac{1}{2}$  thick, they assume a pear-shape in the gall-bladder, and are cylindrical in the ducts, which they frequently much dilate. They have also been found in horses, pigs and cats. Rupture of the gall-bladder has been noticed in oxen, caused by the stone becoming wedged and blocking the outflow of gall.

**Symptoms.**—These differ according to the size and position of the gall-stones. Such as remain in the gall-bladder, or very small stones elsewhere, only cause slight digestive disturbance, and nothing which could be termed illness. They are discovered rather as objects of interest during dissection. This refers to those cases of cholelithiasis in which only one hepatic duct is affected by stony formation. True gall-stone colic, without jaundice, arises when a gall-stone becomes wedged in the bile duct.

**Therapeutics.**—Treatment consists in the use of Carlsbad salts or of alkalis, in regulated diet and plentiful exercise.

DISEASES OF THE LIVER DUE TO PARASITES.—LIVER-FLUKE DISEASE.—DISTOMATOSIS.

**Occurrence.**—This disease occurs most frequently in sheep, and more rarely in cattle. Pigs' livers also often contain distoma. Occasionally they are found in horses, buffalos, deer, antelopes, camels, dogs, cats and rabbits. The disease is produced in sheep and cattle by two kinds of distoma, which occur sometimes alone and sometimes together in the liver bile-ducts and gall-bladder.

1. *Distomum hepaticum*,  $\frac{1}{2}$  to  $1\frac{1}{2}$  inches long, by  $\frac{1}{2}$  to  $\frac{3}{4}$  of an inch wide, leaf-shaped; occurs in sheep and cattle, also in goats and pigs, very rarely in horses, asses, camels, rabbits and cats.

2. *Distomum lanceolatum*, much smaller,  $\frac{1}{2}$  to  $\frac{1}{4}$  of an inch long,  $\frac{1}{15}$  to  $\frac{1}{10}$  of an inch broad, lancet or tongue-shaped; occurs in the above animals, except the horse and ass.

**Natural History.**—The liver-flukes represent the platyhelminths of the group trematoda. They live singly, and for their complete development have to pass through several generations and complicated metamorphoses. The following are the several stages:—The ova are provided with a kind of lid, and pass out of the sheep with their faeces. In four to six weeks, if there be sufficient moisture and warmth (development ceases in winter), they change into embryos furnished with a ciliated skin and a borer in front. These ciliated larvae bore their way into certain snails, little shell-bearing limnae (*limnaeus truncatulus s. minutus*). The geographical distribution of the snail corresponds with that of the *distomum hepaticum*. They are very small, about  $\frac{1}{4}$  in. long, with brown, spiral shells, exist all over the world, are very hardy and live more on land than in water. In this earliest host the embryo changes in fourteen days during summer (three to four weeks in winter) into a sporocyst (germinal bag), with germinative cells, which, by internal elongation, form so-called "redies" (tubes of immature distomata), and from these are directly produced (according to Leuckart)—but sexlessly, according to Thomas—the true distoma brood, or so-called tailed cercaria. They are microscopic creatures resembling tadpoles, which live freely in water, and of which about 1,000 spring from one egg.

These still immature distoma very soon become encysted by attaching themselves to grasses, etc.

Their ingestion by sheep follows consequently in the encysted state in the process of grazing. It may also occur through swallowing the snails or water containing cercaria. Thomas first proved that these tailed cercaria quit the bodies of their first hosts, and, after swimming about a while, fix themselves to water plants or moist grass, where they shed their tails, roll up into a ball in a gum-like matter and thus form snow-white cysts,  $\frac{1}{5}$  inch in diameter. In this state they can exist several weeks, and are eaten by sheep with the grass. Spinola proved by experiment that sheep may also suffer from eating the snails. In the Sandwich Islands, according to Lutz, it is the drinking water which chiefly spreads the infection.

The life history of the *distomum lanceolatum* is up to the present unknown.

**General Remarks on the Occurrence of Distomatosis.**—The existence of this form of disease has been known for more than 500 years. It occurs enzoötically, and even panzoötically, in very wet seasons, which are especially favourable to the growth of the offending snail, but is rare in dry summers. It is consequently frequent in damp districts, or those subject to floods. In 1873 Alsace-Lorraine is said to have lost by this disease one-third of all its sheep, valued at £46,000. In England the annual loss is estimated at one million sheep. In Slavonia, in 1876, 40 per cent. of all horned cattle are said to have perished from the ravages of liver-flukes. From the statistics of the Berlin abattoirs, it was shown that 36 per 1,000 of all cattle, 7 per 1,000 of sheep, 2 per 1,000 of calves and 1 $\frac{1}{2}$  per 1,000 of pigs contained distoma.

The distoma brood is usually absorbed by cattle pastured in damp, swampy places, where the pools and puddles formed by continuous rain are peculiarly dangerous. The lesser spear-wort (*Lysimachia nummularia*), a plant formerly blamed by shepherds, has nothing to do with the matter. Lambs and yearlings sicken most readily, as well as animals of feeble constitution, e.g., fine-wool merinos. Infection may occur in the byre from eating green fodder, or drinking water which contains the distoma. It may even possibly happen with dry fodder.

Occasionally the disease seizes hares and deer, especially

the dams, which latter will waste away to a skeleton and perish. In the East Indies it is quite common among buffalos.

#### IMMIGRATION AND EMIGRATION OF DISTOMA.

The liver-fluke does its work in the warmer months, until the first frosts appear. Night frosts do not seem to harm it, and it may attack as late as December. Observation has proved that animals may very quickly become infested, even after a quarter to half an hour's feeding in an infected pasture. Repeated invasions of the same animal may occur at different times, or of several animals in the same herd at different periods. With regard to the mode by which the creature penetrates to the liver, the following three methods are possible:—

1. By way of the portal system. But proof of this supposition is lacking.

2. After losing through digestion their covering membrane, the distoma may perforate the stomach and small intestine, and, by way of the peritoneal covering of the liver, reach the parenchyma of the same, as well as the biliary ducts (Gerlach, Spinola, May). The occurrence of perihepatitis (peritonitis) has been adduced in proof of this; but it may just as easily arise from centrifugal wanderings of the fluke within the liver, especially beneath the serosa, or from perforation of the latter from within, outwards. Indeed, distoma have not unfrequently been found with their heads projecting from the liver surface.

3. The distoma may wander from the duodenum through the biliary ducts, and thus reach the liver. This supposition (Leuckart's) has most probability in its favour.

The further migrations of the distomum within the liver occur, according to Leuckart, with *distomum hepaticum*, as follows:—The cone on its head serves as an instrument whereby to widen the narrower bile-ducts, while the scale-like points on its fore part prevent any slipping back. Forward movement is procured by alternate application of its oral and abdominal suction-discs, with lengthening and shortening of the fore-part of the body. In *distomum lanceolatum*, which lacks these lateral bristles, progress is easier owing to its smaller size. The greater number of the flukes remain in the bile-ducts; others perforate the walls of the ducts by means of their head-cones and pierce into the parenchyma of the liver, which they

destroy, perforating even the capsule as well, producing perihepatitis, or peritonitis. A third detachment reaches the branches of the portal vein, where it excites endophlebitis, thrombosis and emboli. Finally, a fourth party seems to attain the veins of the liver, and thence the more distant organs of the body. First, after passing the right side of the heart, they reach the lungs, where, as many have observed, they produce haemorrhagic foci, passages charged with bloody matter, and even give rise to nodules in the lungs. In cattle, for instance, cysts or nodules have been found in the lungs, from the size of a hazel-nut to that of a hen's egg, which cysts show thickened and partially calcified walls, within which the flukes float in a brownish viscid fluid. Lung-nodules containing distoma have been found in as many as 10 per cent. of slaughtered cattle. Each nodule held one or two of the flukes (pseudo-tuberculosis). According to Morot, a bronchial distomatosis also takes place, with accumulation of distoma and obstruction or dilatation of the bronchi. From the arteries of the lungs the flukes proceed into the veins, and through them into the general system of arterial circulation, thus reaching every part of the body. According to Willach, the embryos of distoma have some connection with periodic inflammation of the eyes in horses and cattle.

The departure of the liver-flukes takes place when they are sexually mature, which occurs in about three weeks, according to Leuckart. Gerlach asserted that their exit takes place nine to twelve months after entrance, that is to say, from May to July. But Pech and Friedberger noticed great quantities of flukes in autumn and also in winter, located in the gall-bladder and duodenum. In the large intestine they are quickly digested, and therefore never found. Thomas estimates the average life of distoma at over a year.

**Autopsy.**—The most important changes found in cases of distomatosis are, as might be expected, in the liver. They vary much according to the stage of the disease. The liver is much enlarged, twice or thrice its normal weight, its margins blunt and the whole organ discoloured. The colour suggests porphyry, the ground being a dirty grey yellow or greyish brown, upon which are irregularly scattered spots like islands, which are black-red to dark reddish brown in colour, and either round or long in shape, being also sometimes clustered like branches. The upper surface of the liver is rough, uneven and covered with thread-like or membranous peritonitic proliferations, which

frequently cause the anterior surface of the liver to adhere closely to the hinder part of the diaphragm (*perihepatitis*). Not unfrequently holes are seen in the liver-capsule, from a pin-head in size to a millet corn, through which a dirty bloody matter oozes, or the head of a liver-fluke protrudes. If we cut the capsule at this point, we shall often find beneath it a great number of young distoma, and, on their removal, sub-capsular cavities and hollow passages. The lymphatic glands on the hilus of the liver are oedematous, swollen and distended.

On section of the liver, in addition to serious changes of the biliary ducts, several void spaces of irregular shape and dimensions are noticed in the parenchyma, which are partially traversed by ridges containing a bloody, red-brown, or often greyish and pulpy matter, consisting of white and red blood-corpuscles, fatty degenerated liver-cells, waste material and a fluke, usually undeveloped. The biliary ducts are, as a rule, enlarged, often to thrice or four times their normal size. In cattle they may be as much as  $1\frac{1}{2}$  in. in diameter, and the liver thereby seems as though inflated with air. In the ducts the bile is found red and slimy, and there are many distoma with their eggs. In the liver of a sheep as many as 1,000 flukes may be met with. The mucous membrane of the biliary ducts is at times in a condition of haemorrhagic or mucous, or even ulcerous catarrh, owing to irritation by the distoma, and in later stages of the disease is thickened by new growths of connective tissue, often to an extraordinary degree. To this may be added an incrustation of calcium salts (phosphate of lime), which transforms the bile ducts into stiff, unyielding calcareous tubes, which can be felt from the outside and show prominently on the surface. Between these the true substance of the liver is more and more atrophied; it grows harder, creaks when cut and displays a lumpy surface (atrophy of the liver, cirrhosis). In the branches of the portal vein thrombi are sometimes found, produced by distoma moving freely in the vascular lumen. The gall-bladder usually contains a great quantity of muddy, dirty bile, single flukes and often a large number of their ova.

In addition to all these changes of the liver, ascites is nearly constantly observed in severer cases, along with isolated liver-flukes living free in the abdominal cavity, and transuded matter in the thoracic cavity and pericardium. The carcase is very much emaciated, poor in fat, or else the fat is changed to a soft, gelatinous substance; the muscles are lax and pale;

the blood is thin and watery and the subcutis oedematosly infiltrated (hydæmia and anæmia).

**Symptoms.**—1. *In sheep* there is nothing characteristic about these. Indeed at first there are no signs of illness. The minimum duration of the latent stage is from five to six weeks, even with the most feeble animals. When, in the course of 1½-2 months, the changes in the liver have attained a certain degree of intensity, the symptoms which appear are those common to other diseases, such as severe cachexia, successive disturbance of nutrition, as well as hydæmia and hydrops (so-called dry-rot of sheep). The sheep lose condition quickly, soon tire, show pale, washed-out mucous membranes, dry, lustreless wool which soon drops off, oedema on the eye-lids, on the larynx and hypogastrium. In cattle the coat becomes dull and ruffled, and the skin dry and adherent. Then gastric disturbances supervene. Appetite is bad or depraved, rumination ceases, constipation alternates with diarrhoea, the urine becomes acid, etc. Sheep which are thus affected seem very listless and linger behind the flock, can easily be caught, and their temperature shows many sudden and extreme rises. Later follow the symptoms of congestion: ascites, bronchial catarrh, etc.; the conjunctival sac contains an accumulation of mucous secretion.

2. *In cattle* the severer symptoms appear late, and the result is rarely or almost never a fatal one. It has been abundantly proved that in slaughtered beasts a very high degree of liver destruction may often exist without any signs having been noticed in life. (This is the opinion of an expert in forensic cases!) On the other hand, Wiegel asserts that he has not unfrequently seen the disease end fatally in cattle.

**Diagnosis.**—Great diagnostic importance was earlier attributed to palpation to discover enlargement of the liver, and to the jaundiced discolouration of the visible mucous membranes and of the skin. But these signs are lacking more frequently than they occur. Like Gerlach, we have never observed jaundice. Moreover, an enlargement of the liver beyond the posterior edge of the last rib, combined with greater sensibility of the organ, can probably only be demonstrated during the first stage; for in more advanced conditions of the disease we have always missed this sign. Much more important for diagnosis is the microscopical discovery of distoma-eggs in

the faeces (oval and with a lid). In by far the greater number of cases no certain diagnosis is possible without actual *post-mortem*, in which, however, one must not forget that single distoma are extraordinarily common in perfectly healthy sheep and cattle.

**Course.**—This is chronic. Cases of apoplectiform death (embolus on the brain ?) are very rare. According to Gerlach, the progress of the disease may be divided into four stages : 1st, that of traumatic inflammation of the liver—inflammatory swelling of the liver, generally not recognised during life and occurring mostly in the late summer ; 2nd, the stage of chlorosis, six to twelve weeks after pasturing on infected meadows ; chief months, September to November. 3rd, that of emaciation, beginning a quarter of a year after reception of the fluke brood, the severest period of the complaint, marked by atrophy of the liver and formation of oedema ; generally from January onwards. And 4th, the stage during which emigration of the distoma takes place, which Gerlach fixes at May to June, but which may synchronise with the sexual maturity of the leeches, or say three weeks after their entrance.

Where the invasion is very numerous, the course may be correspondingly rapid ; but the minimum duration to death is never less than three months. On the other hand, the signs of chlorosis and wasting may, with older and stronger animals, pass away after a year, and recovery be the result. But this is often only apparent ; permanent changes of the liver in various degrees always remain behind. In general the prognosis in cases of developed distomatosis is very unfavourable, and in this respect the winter and spring months are most dangerous.

**Distomatosis in Other Animals.**—A case in a she-ass is reported by Cadetac. The hepatic ducts were dilated like a bottle (ampulla). The animal grew very thin, showed great paleness of mucous membranes, and, on dissection, revealed ascites, hydrothorax and hydropericardium. According to van Vaezen and Pease, distomatosis shows itself in buffaloes in loss of condition, stiffness of gait in the hind limbs, diarrhoea, tympanites, oedema of the skin and paralysis. It is much more destructive in the East Indies than in Europe. Common salt has been found an efficient prophylactic.

**Treatment.**—As distoma in the liver are inaccessible, their treatment consists, above all, in prophylaxis. The chief

points are, avoidance of the injurious meadows and destruction of the ova. Pastures become more dangerous as the damp weather sets in. As the shepherds' proverb says : " Suspected pastures must not be grazed after the feast of St. John." Care is most needful with lambs and yearlings. It is also important to seek for and destroy the snails in which the larvae develop. Pains must also be taken to keep the sheep well nourished, as they can then better support and resist the injuries inflicted. Thomas recommends prompt slaughtering as the best means of destroying the ova, and this is perhaps the wisest course. The livers should be buried, or destroyed by heat ; but when boiled may be safely given to dogs to eat. The affected animals and their droppings should be removed to dry pastures ; wet fields should be drained and sprinkled with salt or lime ; and rock salt should be supplied for the sheep to lick, in order to kill such immature distoma as have already reached the stomach. Such provision of salt may entirely supersede the bitter, aromatic or astringent remedies formerly employed. According to Frasbol, good results have followed from feeding the sheep with pine-tree shoots mixed with clover before driving them out to pasture. If the enzootic spread of the disease cannot in any way be checked, then the only thing to do is to cease keeping sheep in the infected district. To prevent the introduction of the disease by strange sheep, Leuckart recommends the examination of their dung under the microscope, to see if it contain any ova of the fluke. Thomas says that sound meadows may possibly be infected by hares and deer.

The period of warranty is in Bavaria and other parts of central Germany fourteen days, which term varies in different parts of the German Empire from 14 to 42 days and in Austria is two months.

**Other Distoma.**—Besides the *distomum hepaticum* and *lanceolatum* the following occur : *Monostomum hepaticum* in the livers of swine; *distomum truncatum*, *albidum* and *felinum* in dogs and cats; *distomum magnum* in Texas cattle, in stags and antelopes; *distomum pancreaticum* in the cattle of Japan; and *distomum heterophyes* in the dogs of that country.

**Distomatosis of River Crayfish. Distomatosis astacina.**—The so-called "cray-fish plague," which has for some years infested the waters of Central Europe, has been traced by Hartz to the incursions of the larvae of the *distomum cirrigerum*. Its symptoms are high, stiff gait, a loss of all power of resistance, swelling of the anal orifice, loss of sensation and irritability, twitchings, paralysis. On dissection, the

larvae are found encysted chiefly in the muscles. This distomatosis must not be confounded with other crayfish plagues (*mycosis astacina*, caused by decayed wood-saprolegniae) or with poisoning.

## II.—ECHINOCOCCUS DISEASE.

**General Remarks.**—This disease is caused by the ingestion of *echinococcus polymorphus* (formerly *echinococcus veterinorum*) (the bubble or cyst-worm), which is the larval stage of *tænia echinococcus* in the dog. It affects most frequently cattle, and after them, sheep, goats and pigs; most rarely the horse, ass, cat, rabbit and dog; but also men, camels and various wild ruminants (deer, chamois, antelopes, etc.), and finally apes, various animals of the cat tribe, turkeys, etc. The *echinococcus* cysts have been found in almost every organ of the body, but their chief seats are the liver and lungs. They also occur in the heart, spleen, kidneys, the muscular system, the oesophagus, in omentum and mesentery, beneath the serous membranes, in the eye, brain, udder, and even in the bones themselves. *Echinococcus* sickness is pretty widely distributed. In South Germany it is said (Bollinger) to be the commonest of all infectious diseases among ruminants. Its geographical area corresponds with that of the dog. According to statistics compiled by Peiper from 52 slaughter-houses all over Germany, 11 per cent. of all cattle killed, 10 per cent. of sheep and 6½ per cent. of pigs suffered from this complaint. In some districts he says that the percentage was as high as 65 per cent. in cattle and 51 per cent. in sheep. In British India, where dogs are largely kept by the lower classes, *echinococci* are found in the livers of 70 per cent. of all cattle.

**Natural History Notes.**—The *echinococcus* cysts, which represent the larval stage of the three-segmented tape-worm of dogs, develop after consumption of *tænia* ova in the food, water, etc. They appear in four different forms—first, and most frequently, as a so-called *simple echinococcus*, consisting merely of a capsule, filled with fluid and containing brood-germs. Second—as *amalgamated echinococci*, in which the principal cyst is broken up into so-called "daughter" (or secondary) cysts, or even into "grandchild" (or tertiary) cysts, which spring partly from the outer surface (exogenous), and partly from the inner surface (endogenous) of the parent cyst. Third—as so-called *echinococcus multilocularis* or *alveolaris*, a

new growth resembling colloid cancer, which displays lacuna spaces on section. This new growth seems to have arisen from the expansion of an echinococcus which has no capsule, or has broken through its capsule, and develops mostly in the hepatic canals, and later often closely resembles conglomerated tubercular nodules. It is sometimes as hard as stone, but this occurrence is rare. Fourthly—*echinococcus acephalus* (acephalocysts), which are headless and sterile cysts; they are very common.

The development of echinococci from the embryos, which probably wander through the biliary ducts and portal vein system, is rather slow. In four weeks the cyst is  $\frac{1}{8}$  inch long, in eight weeks  $\frac{1}{6}$ — $\frac{1}{5}$  in. long; after nineteen weeks it has attained the size of a nut, and only after five months do the brood-capsules, containing the heads, begin to show. The true wall of the cyst is gelatinous, of lamellated structure, and consists of an outer and inner cuticle containing chitin. Around this gelatinous capsule is a solid capsule of connective tissue, which consists of granular matter, sparse muscular fibres and vessels. The echinococcus cyst, which ranges in size from a pea to a man's head, is filled with clear serous fluid, free from albumen. The largest ones hold as much as 10lbs. in weight. The scolices develop from the germ capsules (brood-germs) in the wall of the cyst, each of which may contain as many as 22 scolices, so that often one bladder produces 1,000.

**Echinococcus multilocularis.**—This is in general rare, but occurs, as Ostertag has shown, more commonly in cattle than was supposed. During one year he found it 30 times in cattle in the Berlin abattoirs (among 200,000 beasts), only once in a pig and not at all in sheep. The E.m. of men is distinguished from that of cattle by the fact that the latter causes no clinical symptoms. Ostertag considers it probable that the multilocular E. does not represent a mere variety of the ordinary E., but a particular species with an as yet unknown tapeworm. This idea has been confirmed by recent investigations of Müller and Mangold. Indeed, two sorts of *tænia echinococcus* are actually present in dogs. The common *tænia E. unilocularis* has plump hooks, and the *tænia E. multilocularis* much thinner ones.

**Autopsy.**—Livers affected by echinococcus cysts are usually more or less enlarged, sometimes even five or tenfold, and present a lumpy surface, corresponding to the cysts. Their weight is also proportionately increased. Thus a liver, whose normal weight is about 10lbs. may be 150, or even 158lbs. in weight. One such, weighing 132lbs., contained 2,400 cysts.

A pig's liver of 4lbs. may grow to 50 lbs., and Perroncito found one of 110lbs. The serosa of the liver is often thickened, and the liver itself firmly adherent to the neighbouring diaphragm, omentum or intestines (perihepatitis). The echinococcus cysts dislodge the parenchyma of the liver, so that the latter is found on section, to be permeated by a number of cavities, between which compressed remnants are left, which are atrophied, insulated or striated. If the cysts die away, their contents change to a yellow, fatty, greasy and pulpy mass, which often seems to consist of chalk only. Pus may also be formed therein; blood occurs only rarely. The whole can then only be distinguished microscopically by the discovery of the characteristic hooks of scolices.

The echinococcus cysts grow in the lungs to a size varying from that of a pea to a human fist. There also they produce the same lumpy exterior and compression of the parenchyma, so that often only a small part of the original lung-tissue is left, while the compressed parts are extremely hard. Uhse compares such a lung to a "leather sack full of potatoes." The normal 6lb. ox-lung may weigh as much as 50lb. In one case Becker found 2,300 cysts in the lungs of an ox, and 5,000 in the entire carcase. When the cysts die out, a caseous, degenerated detritus is produced, which looks much like old nodules of tuberculosis.

In the heart only one such cyst is generally found, and this mostly at the lower end of the septum, near the apex, and may enlarge either towards the ventricles or towards the pericardium. Echinococci occasion sometimes tumour-like swellings in the bones, e.g. in the posterior maxilla; also in the medullary cavities of the tibia and the femur. They are, moreover, not uncommon in the muscles, particularly of pigs.

**Symptoms.**—These vary according to the organs affected, but are in general not very characteristic. Cattle particularly may appear outwardly quite healthy, in spite of the presence of echinococci in large numbers. As many as 2,400 and 2,600 have been found in the livers of slaughtered cows, and 5,000 in the whole body, which showed no signs of the disease when alive (Becker, Posnjakow).

I. The chief signs by which they may be located in the *liver* are: disturbed digestion, gradual wasting and a general sickliness lasting for years. Jaundice is rare. The liver shows itself on percussion to be more or less enlarged on the right

side in the region of the last two or three ribs, that is to say, from the tenth or ninth rib to the posterior edge of the last but one. Palpation proves the liver region to be sensitive. Sometimes a tumour can be seen in the neighbourhood of the right flank, or lumpy, fluctuating irregularities felt upon the liver, on examination through the rectum. Fever is usually absent, or occurs only with other complications, or in the last stages of the disease. When the superficial cysts burst their contents pour into the abdominal cavity, where they may produce fatal peritonitis (rare).

2. The invasion of the *lungs* by echinococci causes (like tuberculosis) the aspect of pulmonary consumption, with gradually increasing emaciation, hardness of the skin, etc. The illness manifests itself in growing difficulty of breathing, which, when the invading echinococci are numerous, may develop with extraordinary rapidity, when the breathing becomes a series of interrupted gasps, and a weak, harsh cough occasionally sets in. On percussion one obtains, according to the position of the cysts, a deadened valvular sound (rarely as though empty), the sound of the broken vessel (*olla rupta*), as well as an increased resistance under the hammer. On applying the ear or stethoscope, the normal vesicular breathing sound has vanished, and strange murmurs are heard—according to Harms, a squelching noise (the same sound that is heard on pressing or kneading a section of lung containing echinococci, and which Harms considers pathologically important). Also humming and cooing noises are perceived and (according to Schmidt) a peculiar metallic ring. On striking more forcibly on the thoracic wall, the beast retreats and groans, particularly if struck in the region of the last four ribs. Fever is generally absent. Appetite and yield of milk may for a long time be fully maintained. If a cyst be penetrated in the thoracic cavity, pneumo-thorax with fatal pleuritis may be developed.

3. Echinococcus cysts in the *heart* sometimes produce death resembling apoplexy by bursting or inducing paralysis of the organ. In such cases the animals drop suddenly and die, or exhibit symptoms of heart-weakness and suffocation.

4. In the *muscles* the cysts may cause lameness. Johne found one such in the psoas muscles of a horse.

**Prognosis.**—Whenever a large number of cysts are present in any one organ, this is unfavourable, and death may result. But single echinococci may become caseated or calcareous, and a great many are often found in the lungs and

liver of otherwise quite healthy animals. Yet for the heart a single echinococcus may prove very dangerous. If once the disease can be identified, even though it be only approximately, the wisest course is to have the animal slaughtered.

**Diagnosis and Differential Diagnosis.**—This disease is by no means easy to identify. When in the *liver*, we must first of all demonstrate an enlarged liver-dulness, sensibility to pain in that region, dilatation of the organ, etc. Also we must discover whether the liver possesses a lumpy surface, by examining it through the rectum. But these signs may be confused with those of pregnancy, hydrometra, pyometra, new growth in the womb, ovarian tumours, abdominal pregnancy, all of which are excluded by rectal exploration; also with ascites, which may be known by percussion and by the changed conditions in the dorsal position; and finally with other, and especially cancerous liver-tumours, which, however, are of rarer occurrence. Also one might be in doubt as to whether it were a case of hydro-nephrosis.

When the disease is in the *lungs*, it is chiefly important to differentiate it from tuberculosis. According to Harms, the following are the signs to note:—1. In echinococcus sickness there is no cough, or it is very weak, whereas it is never absent in tuberculosis and is comparatively strong. 2. The squelching or croaking sound is in echinococcus sickness pathognostic. 3. Breathing is also more rapid and difficult in the last named sickness, and the difficulty increases more quickly, and often, as compared with the animal's external condition (generally quite normal), this increase is startlingly quick. 4. Friction fremitus is absent in hydatid disease, whereas in tuberculosis it is frequently (?) audible. 5. Of importance in echinococcus sickness is its complication with demonstrable enlargement of the liver. Moreover, in tuberculosis of the lungs one frequently finds a co-affection of the lymph-glands as well as tubercular inflammation of the udder.

**Treatment** is of no avail. The utmost possible is, as in men, to attempt puncture of all accessible echinococcus cysts. On the other hand, prophylaxis is of the greatest importance. This consists in destroying the cysts, which indeed ought to be legally obligatory, seeing their great danger to men and their economic injuriousness; also in ridding dogs of *tænia* and a reduction in the number of dogs by heavy taxation.

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**Echinococcus Sickness in Man.**—This is by no means rare, and is of importance to veterinary surgeons because they are peculiarly subject to infection by *tænia echinococcus* when treating dogs for tape-worms. In Central Germany 5 per 1,000 deaths are traceable to this disease; and in Iceland from 5 to 10 per cent. of the population perish from it. The symptoms develop slowly. They consist in the formation of a fluctuating, soft, palpable and lumpy liver-tumour, with pressure and a feeling of tightness on the right side, dyspnoea and bronchial catarrh caused by pressure of the liver upon the diaphragm; atrophy of the liver, jaundice, with occasional formation of abscesses internally or externally. If located in the lungs, the cysts may (though only rarely) be coughed up, and the disease thus recognised. If in the kidneys, we find tumour there and pain round that region, and a few of the hooks in the urine. A cyst in the brain causes local symptoms. Death occurs, on the average, after five years. Peiper has shown that the frequency of this disease in man is proportionate to its occurrence in animals. It is commonest in Mecklenburg and Pomerania.

### III.—COCCIDIOSIS IN RABBITS. GREGARINOSIS.

**Occurrence.**—Coccidia, especially *coccidium oviforme* in the liver, that is in the bile-ducts and intestinal canal, occur en-zootically in summer in young rabbits. They have also been found in the same organs of dogs, calves, pigs and sheep, also in birds, amphibia, fish and in man. The disease seems to be connected with the food supply in certain districts. It is moreover transmitted to young rabbits by older ones suffering from liver-coccidiosis.

**Natural History.**—According to Pfeiffer coccidia belong to the group of *sporozoa*, which are monocellular, low protozoa, propagated by the formation of spores, and standing between the animal and vegetable kingdoms. Among these sporozoa three orders may be distinguished as important from the point of view of animal pathology:

1. Coccidia, whose chief form is *coccidium oviforme*. This latter lives in the epithelium of the bowels and liver (bile-ducts) of rabbits, where it produces so-called coccidiosis of the liver (formerly known as liver-gregarinosis of rabbits). It also causes enzoötic catarrh of the respiratory mucous membrane. Coccidiosis of the bowels also occurs in birds as well as in mammals, and is produced by a *coccidium oviforme*. Other kinds are *C. perforans* and *bigeminum*.

2. Sporidia (*psorospermia*, *sarcosporidia*) which live as parasites in the muscle cells of mammalia and birds, and also in the blood-cells (malaria, Texas fever).

3. The *Gregarines*, viz : those living in swarms, very highly organised, naked sporozoa.

The *Coccidia oviforme* (egg-shaped), formerly designated gregarine or psorospermia, are, when fully developed, egg-shaped objects with double contour, closely resembling oxyuris ova, and measure on an average  $\frac{1}{100}$  by  $\frac{1}{150}$  inch. They settle in the epithelium of the mucous membrane of the bile-ducts and bowels. They are enclosed in a firm, solid, transparent shell, and contain a coarse-grained protoplasmic substance. They appear to multiply wherever they lodge, but especially in the liver, by formation of spores, and also outside the body in the open. The contents of the coccidia break up into small, egg-shaped spores, which excrete a sickle or kidney-shaped transparent object, which strongly refracts the light. These are to be regarded as the young of the coccidia, which assume a globular shape without visible organization, and effect amoeboid movement by extension or retraction of processes.

**Autopsy.**—In gregarinosis the rabbit's liver is enlarged and sometimes has a lumpy surface. Its parenchyma is permeated by whitish-yellow nodules or cysts, in size from a millet-grain to a bean or hazel-nut, which sometimes lie so close together that the liver-tissue beneath is completely hidden. These nodules, which often closely resemble tubercles or other new growth, are to be regarded as parenchymatous proliferations of the mucous membranes of the biliary ducts, and contain a thick, yellow liquid, in substance creamy, crumbling or caseous. Under the microscope they are seen to be made up of capsules, egg-shaped coccidia, fatty-degenerated epithelial cells, loose nuclei, drops of fat, etc., and separated from the parenchyma of the liver by a thick capsule of connective tissue, with numerous nuclei and fibres concentrically arranged.

In the intestinal canal of young rabbits coccidia are found lodged in the epithelial cells; also in dogs, sheep, pigs, cats, poultry, etc. Sometimes they lie singly, at others in groups, when they form small, elevated, whitish spots on the intestinal membrane. Finally they may even cover larger areas of the mucous membrane in the form of pseudo-membrane. They are most numerous in the intestinal villi, which stand out as white spots; also in Lieberkühn's glands, whose surroundings sometimes show inflammatory infiltration with formation of ulcers. The presence of coccidia leads moreover to inflammation of the intestinal mucous membrane.

## DISEASES OF THE LIVER DUE TO PARASITES. 263

Ralliet and Lucet observed a coccidiosis of the kidneys in a goose (*coccidium truncatum*). In the kidneys were numerous nodules the size of a pin-head, and free coccidia in the tubuli uriniferi.

**Symptoms.**—In the case of young rabbits, sometimes an entire warren is swept away, and the symptoms consist in diminished appetite, yellow discolouration of the mucous membranes, discharge from the nose, weakness, wasting, exhausting diarrhoea, tympanites, staggering, etc. Death ensues with convulsions. But in older rabbits no visible symptoms manifest themselves, in spite of the presence in the liver of considerable numbers of coccidia.

**Differential Diagnosis.**—Mistakes in diagnosis were formerly common. Hake, who first discovered the coccidia, thought they were pus corpuscles, and Lang took them for new growths. They are most frequently confused with the larvae of pentastomes, tapeworms and oxyurides (Willach). Concerning psorospermious utricles, which in literature are often treated along with coccidia, we must refer our readers to the chapter on muscular diseases (Vol. II., chap. XI.).

**Therapeutics.**—In treating liver or bowel gregarinosis only prophylaxis is of any avail. This consists in separating the sick animals and disinfecting their stalls. Of internal remedies, at the utmost, a mercurial preparation, perhaps calomel, might be tried as an anti-parasitic drug.

**Other Parasites of the Liver.**—In rare cases we also find in pigs' livers *cysticercus tenuicollis* and *pisiformis* (measles) and cysts of *taenia marginata* and *T. serrata*. These have also been found numerously present in the lungs as well as liver of two pigs.—Megnin observed a case of chronic hepatitis in horses, caused by *sclerostomum armatum*. Liver complaint is said by Dinviddie to occur in Arkansas as the result of *stephanurus dentatus*.

## CHAPTER V.

### DISEASES OF THE PERITONEUM.

#### ACUTE PERITONITIS.

**Classification.**—Peritonitis may be classified from different points of view. It is first of all advisable to divide it into the acute and chronic forms, and this on purely practical grounds and in view of the course of the disease. Further, according to its distribution, we might define a circumscribed and a diffuse form; or according to its origin, a primary or idiopathic, a secondary and a traumatic; we might speak of perforative peritonitis, tuberculous, sarcomatous, carcinomatous, pyaemic, metastatic, septic and rheumatic peritonitis. Further, according to the nature of the products of inflammation, we distinguish a dry and exudative, a serous, sero-fibrinous, ulcerative, haemorrhagic and an ichorous peritonitis. As regards origin, we generally have to deal with a secondary inflammation of the peritoneum, caused either by injury or by pathological processes in the adjoining parts. Yet the possibility of idiopathic peritonitis, resulting from previous chill (rheumatic), cannot be disputed. But such cases are rare, for although rheumatic colic is fairly frequent in horses, yet rheumatic peritonitis occurs only in isolated instances.

**Occurrence.**—Acute peritonitis occurs in all domestic animals, including poultry. Females are, however, more predisposed to it than males, which may be explained by the closer connection existing between their organs of generation and the peritoneum. Thus, in cattle, we meet the complaint much more commonly than in horses, because they are more frequently pregnant and are more often exposed to inflammation, rupture, etc., of the uterus during the act of delivery, from which peritonitis usually results. Moreover, in cattle,

peritonitis not unfrequently follows as a complication of distomosis of the liver, peritoneal tuberculosis, etc. The peritoneum of domestic animals differs singularly in sensitiveness. That of the horse seems to be most sensitive, then follow the ruminants (sheep, goats, cattle), next the carnivora (dogs and cats), and last of all the omnivora (pigs) and poultry.

**Etiology.**—The causes of acute peritonitis are as follows:

—1. Penetrating abdominal wounds, both accidental and such as result from operations. Under the latter head come all those caused by opening the abdominal cavity (laparotomy), such as herniotomy, puncture of the bowels or abdominal cavity, opening of the paunch (enterotomy), castration of the female animal and castration of subjects with undescended testes. The true cause of the inflammation is to be sought in the introduction of irritant substances (bacteria) by means of the air, or by insufficiently disinfected instruments. Thus it is quite possible for the inrush of air through the inguinal canal to set up, under certain circumstances, fatal peritonitis during the castration of a stallion.

2. Rupture and perforation of organs covered by the peritoneum, with consequent intrusion of irritant matter into the abdominal cavity. Among these are ruptures of the stomach, bowels, urinary bladder, liver, spleen, etc.; laceration and perforation of the walls of stomach or bowels by ulcers, foreign bodies, tightly-wedged faeces, and intestinal worms (*gastrus larvæ, ascarides*); rupture of the womb during parturition; rupture of the gall-bladder; perforation of the liver-capsule by distoma; penetration by abscesses, *echinococcus-cysts*; rupture caused by thrombosis of the vessels in haemorrhagically-congested and narcotic bowels; perforation of the rectum during examination.

3. The spread of inflammation from an adjacent organ to the peritoneum, as, e.g., from the abdominal walls after contusion; from stomach or bowels after intense inflammation of their mucous membranes; from ulcers, partial ruptures, displacement of intestines (twisting, invagination, incarceration, strangulated hernia); further from inflamed condition of the liver, kidneys, bladder, womb (metritis, para and peri-metritis), of the ovaries, testicles or of the stump of the spermatic cord; and finally, though less frequently, as a result of previous pleuritis.

4. Chills, whether external of the abdomen, or internal by

absorption of cold drinks. According to Trasbot, it may also arise from cold injections into the rectum. This so-called rheumatic peritonitis seems, however, to be relatively rare.

3. On the occurrence of metastatic peritonitis in the course of pyæmia and septicæmia in our domestic animals little is as yet known.

Investigations have, in late years, been made into the micro-organisms which produce peritonitis. Predöhl assigns an important rôle to *streptococcus pyogenes* in the production of peritonitis, particularly of the traumatic form. Bumm differentiates between a streptococci-peritonitis (pus) and a putrid form (ichor), as well as a combination of both. The *bacterium coli commune* seems to be pathogenically important in perforative peritonitis (Ziegler). Death seems in this case to result less from the peritonitis, i.e., from its resultant septicæmia, than from poisoning occasioned by absorption of poisonous toxines from the faeces, and perhaps partly from nervous shock.

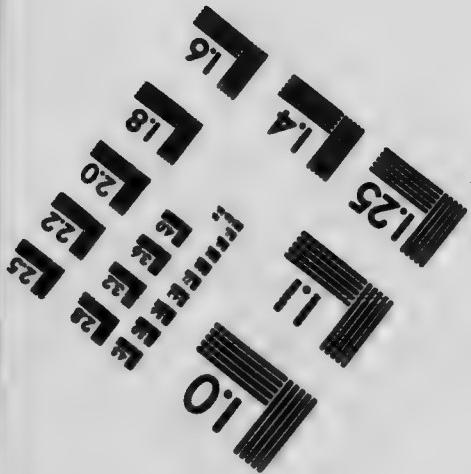
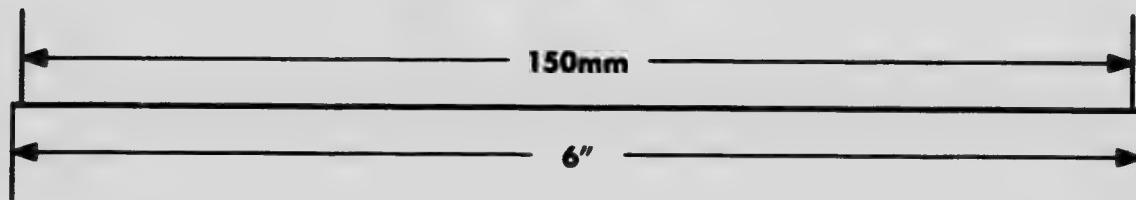
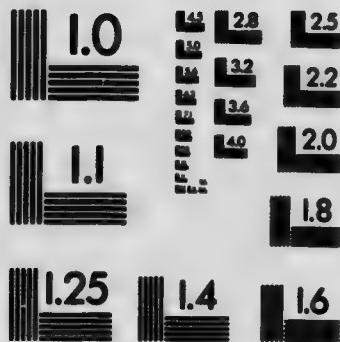
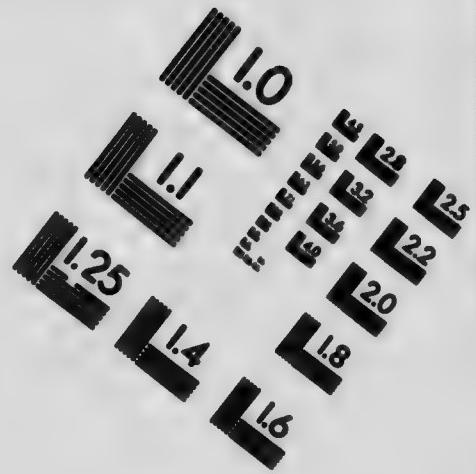
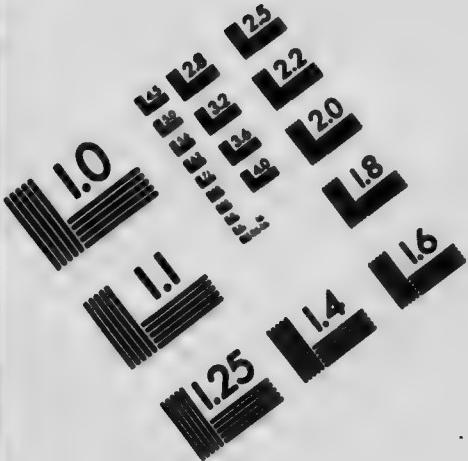
**Post-mortem Lesions.**—In acute diffuse peritonitis the peritoneum is found at the beginning of the inflammation to be hyperæmic and infiltrated by numerous minute dots of extravasated blood. The redness is of varying degree, sometimes uniform and at others irregular; its tint ranging from pink to brick-red and scarlet, at times streaky or spotted. The inflammatory injection is usually not equally advanced in all parts, and its starting point can often be traced. In order to see the hyperæmia of the peritoneum it may sometimes be necessary to wipe away the exuded matter lying over it. The redness diminishes later, and the peritoneum appears a greyish red. The upper surface of the serosa loses its lustre quite early, becomes less transparent, rough like velvet, and the peritoneum itself easier to tear, and detachable. Newly-formed vessels also soon appear in the inflamed tissue. On the inner surface of the peritoneum an exudation, which varies much in form and quality, is invariably deposited and generally soon after the disease begins. This sometimes consists only of a thin layer of coagulated fibrin in the form of a fine, translucent and easily removable membrane. In other cases this deposit is stronger, resembling a yellowish white or deep yellow croup-membrane. By means of this overlying exudation a part of the visceral layer often becomes attached to the parietal layer of the peritoneum; or different organs in the abdominal cavity may be fastened to each other by their visceral peritoneal covering (*peritonitis fibrinosa* or *sicca*). In contrast to these dryer products of inflammation, a large or smaller amount of

yellow to yellowish-red fluid collects in the abdominal cavity in serous or sero-fibrinous peritonitis. This is at times greenish yellow, very turbid, and may, in horses and cattle, amount to eight and a half gallons; it consists chiefly of serum, flakes of pus, fibrin, coagulated and cellular matter. If such exudations contain a large amount of red blood-corpuscles, the fact is shown by a brighter red colour, as has been noted especially in horses (complication of sero-fibrinous with haemorrhagic peritonitis). In cases where there is a perforative peritonitis proceeding from the intestinal canal, this exudation is ichorous in appearance and very foul-smelling (ichorous peritonitis); it is usually also mixed with extravasated contents of the bowel. After rupture of the bladder it has a strong smell of urine. In cats and dogs the exudation in the abdominal cavity is more purulent in character (*peritonitis purulenta*), and either rests on the peritoneum as a thin, creamy layer, or gathers in the abdominal cavity as a dirty yellow fluid, not seldom mixed with fine flakes and smelling very badly, especially after the breaking through of abscesses.

In the later progress of the peritonitis the exudations just named are partially or entirely re-absorbed. To this process the cellular elements and fibrin offer most resistance, as they must first undergo fatty degeneration and become crumbled into fine detritus. After resorption of the fibrinous exudations there often remain adhesions of the peritoneal layers (*peritonitis adhaesiva*), and cords or bands of connective tissue are formed which, by the contraction of cicatrisation, may cause stretching and shrinking of the organs of the abdominal cavity, as well as constriction of the canals (bowels, biliary ducts), etc. If the resorption of the fluid exudations be only partially completed, and a permanent issue of white blood-corpuscles take place, the fluid obtains a purulent and at times a mucous-purulent appearance. In rare cases, if there be partial adhesion simultaneously with a discharge of small particles of exuded matter, these become with the progress of resorption more and more thickened, and transformed into a caseous and even calcareous mass. If the peritonitis last long, the intestine also shares in the process, in so far that, by the occurrence of a collateral inflammatory oedema, its walls are thickened, rendered very moist and may become very opaque, resulting in paralysis of the muscular apparatus of the bowel, and of the entire bowel-section, with stoppage, accumulation of gas, etc. The mucous membrane of the intestinal canal also is catarrhally inflamed. Finally,

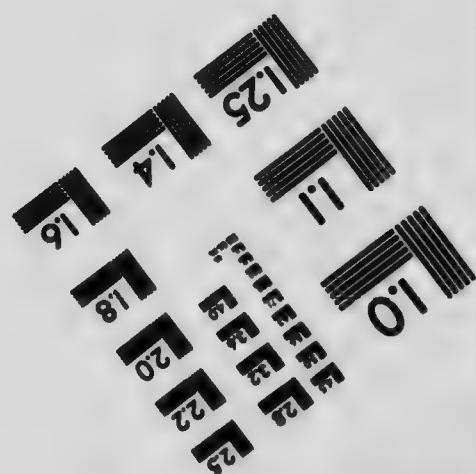


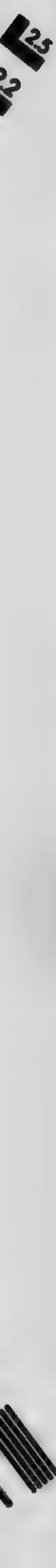
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great quantities of peritonitic exudate may produce injury by purely mechanical pressure upon the diaphragm and other organs of the abdominal cavity, as well as softening of the outer surfaces of such organs. In particular, infiltration of the diaphragm very quickly produces paralysis of this muscle and leads to consequent great difficulty of breathing (*dyspnaea*).

Acute circumscribed, or local peritonitis shows in general the same pathological changes, but they are restricted to smaller portions of the serosa, e.g., to the serous covering of an intestinal part, of the liver, womb or ovaries. Such a peritonitis follows on wounds in the peritoneum (puncture), or on inflammation of any of the organs covered by it (*peri-metritis, peri-hepatitis*). The point of chief importance lies in a local restriction of the irritant matter by early cohesion of the inflamed layers or discharge of the exuded matter. Slighter cases of circumscribed fibrinous peritonitis occur very often in our domestic animals, especially horses, as is not unfrequently proved by the traces of old disease found on dissection.

**Phenomena of Acute Diffuse Peritonitis, with especial regard to Horses.**—These can in no wise be termed typical or uniform. This arises from the fact that peritonitis is, except in rare cases, a secondary complaint, and its symptoms may be changed in many ways by the primary disease, or even completely obscured. Even when the occurrence of peritonitis can be positively established during the course of the primary ailment, it is difficult, if not impossible, to separate the conditions of the two diseases during their further progress. Indeed, the primary disease may run its course with such terrible acuteness, that the symptoms of peritonitis may never come to light, as, e.g., in displacement of the intestines in horses. Moreover, the course varies so greatly in the different kinds of peritonitis. Sometimes it is slow and continuous, and at others the symptoms, from the very beginning, are severe and violent, and progress so rapidly that no special symptom can be singled out. Thus in perforative peritonitis death occurs in a relatively brief time, and yet only the symptoms of sepsis can be identified. The most important signs, and those which may be regarded as especially belonging to peritonitis, are the following :—

1. *Acute pain* shown mostly by lighter or severer attacks of colic. This is the first sign. The colic is often most severe at first and abates later, or shows remission. Thus in a case of

traumatic peritonitis, proceeding from the spermatic cord, we have seen the pains appear quickly and very violently. They manifested themselves in grinding of the teeth, gnawing, snorting, grinning, plunging the head into the manger, spasmodic stretching of the extremities, rolling half-over on to the back, and then avoiding all movement. These signs were accompanied by partial and then profuse sweating. The pains decreased continually during further progress of the disease, or became worse again after temporary abatement. In other cases the phenomena of restlessness are from the commencement to the end only very slight, and are restricted to frequent lying down and groaning, or even at times to mere pawing of the ground, looking backwards, drawing in the back, kicking up against the abdomen and lashing the tail. In a few cases there seem to be no indications of pain whatsoever.

2. The *fever* is generally intense. In most cases of diffuse peritonitis we have noticed temperatures of from 41 to 42° C. (105.8 to 107° Fahr.). With perforative peritonitis the fever may increase very rapidly, accompanied by shiverings. The temperature is moreover very irregular, as is also the course of the disease, and it is impossible to establish any definite fever-type. Its course is usually intermittent or remittent. In severe septic forms of peritonitis the temperature may even be subnormal. The pulse is almost always distinguished by an extraordinary acceleration out of all proportion to the bodily temperature, its speed with horses being often 80 beats and more per minute, rising even to 130. Characteristic of peritonitis is also the fact that at the beginning the pulse is, as a rule, small and hard, growing smaller with the progress of the disease, until it becomes quite imperceptible. The artery is very much contracted and somewhat resembles a wire. As the pulse grows more rapid and feeble, the heart palpitates more and more. The visible mucous membranes, especially of the head, are sometimes more highly reddened or cyanotic, and again grow pale. The superficial temperature is irregular, and the extremities in particular are cool.

3. The *abdomen* is at first very much drawn up, and more or less sensitive to pressure. In simple cases the pain is at first localised, whereas later it is more widespread as the process becomes diffuse. In severer cases this pain disappears with the progress of the disease. Not unfrequently no sign of pain can be found on pressing the abdomen, which may be explained by the much greater difficulty of applying the test of palpation to

a horse, owing to its larger size as compared with other animals, as well as by the fact that it is not easy to examine this part in horses which are already sensitive and ticklish. The abdomen also often becomes visibly enlarged quite early in the disease. The flanks are first distended by accumulations of gas in the intestines, and the tympany may become severe. Then, too, a distension of the abdomen downwards and sideways is noticeable in the smaller domestic animals (less so in the larger ones), caused by accumulation of exudate, which fact enables us to detect, both by palpation and auscultation, the same symptoms as we shall describe in connection with ascites. The peristaltic action of the bowels is completely suspended, often even in the first days of illness, but especially in its later course (paralysis of the bowels by oedematous saturation of the muscular apparatus). In consequence of this, defecation is delayed or entirely stopped, though the animal will often strain hard to evacuate. The dung is generally in small balls, dry and coated with a crust of mucus, and readily collects in great masses in the rectum ; yet severe diarrhoea may set in towards the end. One often notices a certain difficulty in urination, which is shown by the animals frequently placing themselves in position to micturate. In prolonged pericystitis the urine may even be retained for several days (paresis of the sphincter). The passage of water, as well as of faeces, is rendered difficult, owing to the pain caused by contraction of the abdominal walls. As a rule very little urine is passed ; and in proportion to the time that the animals have fasted, it is generally acid, of high specific gravity and usually contains albumen. In one case we found, shortly before death, a great quantity of bacteria in the urine. In peritonitis following on rectal rupture there may also develop extensive subcutaneous emphysema (Friedberger).

4. *Food consumption* may, according to circumstances, entirely cease, or gradually diminish. Later, it is mostly stopped entirely, as well as that of water. As in man, so also in the horse, eructation and vomiting are noticed, though only rarely, which may be traced to an affection of the serosa of the stomach with paresis of the muscular apparatus of the sphincter.

5. *Breathing* is usually accelerated, now short and superficial, and then very deep and laboured, arising not unfrequently to a degree of intense dyspnoea. The causes of this dyspnoea are to be found in a paresis of the diaphragm, consequent on sympathy with its peritoneal covering ; in restriction of space in the abdominal cavity produced by the exudations therein

and by the tympanically dilated intestines; in the painful contraction of the diaphragm and abdominal walls; as well as in disturbances of circulation connected with lowered activity of the heart. The respiration varies from 20 to 70, and even more, per minute; and this action is often entirely thoracic, owing to the paresis of the diaphragm and abdominal walls (remarkable rise and fall of the ribs, and striking participation of the intra-costal muscles). It is said that in cases where the diaphragm is affected, singultus has been observed along with gaspings which shook the whole body; moreover, on palpation, great sensitiveness was found along the insertion of the diaphragm.

6. The *general condition* is, as a rule, greatly embarrassed. After disappearance of the colic attacks, great apathy and sleepiness are shown, combined with debility. The animal stands with hanging head, or leaning against the wall (one that we observed remained standing until death), takes no interest in what is going on about it, shows great weakness, tremblings, local muscular twitching, a stiff and constrained posture, is difficult to move and sometimes neighs for hours before the fatal end. When once it has fallen down, death, as a rule, occurs speedily.

**Course.**—The course of acute peritonitis is in most cases unfavourable. The causes of death are generally found to be sepsis, i.e., poisoning, with great weakness and paralysis of the heart; also at times in suffocation. Death often occurs rather early, especially in cases of perforative peritonitis, in which the end sometimes comes in twelve hours, and may on an average be expected within 20 to 30. In less acute cases the disease may last from eight to fourteen days, and may later pass into the chronic form.

**Acute Peritonitis in Cattle.**—All reports agree that in cattle peritonitis presents symptoms so lacking in definite character that they are often very hard to recognise. Above all, the accumulation of exudations in the abdomen can only in exceptional cases be physically proved, and palpation only rarely reveals signs of pain. Hence it is, that the pulse, which in horses, is so helpful an indication, is in cattle hardly any guide, being under normal conditions so irregular. The symptom of tympanites is also of little importance, as it occurs so frequently in other cases. In general, the appearances which may be noted are: an all-round increase of circumference of the belly, dull manifestations of pain, sensibility to pressure upon the walls of the abdomen, shiverings, constipation and tympanites, constrained movement, stiff postures,

inefficacy of the strongest purgatives, frequent and small pulse. The duration runs from a few days to a few weeks, and the end is fatal in about half the cases.

**Acute Peritonitis in Dogs.**—In dogs and smaller animals the disease is easier to diagnose, because they can be more thoroughly examined by palpation. It is sometimes marked by signs of pain, sensibility of the abdomen to pressure, clearly recognisable exudations in the abdominal cavity, constipation, tympany, stiffness of the hind limbs, groaning during evacuation, dyspnoea and fever. In *Cats* excessive tympanites is occasionally noticed.

**Acute Peritonitis in Poultry.**—This arises from perforation of the stomach or bowels by foreign bodies, from rupture of the Fallopian tube, parasites in the abdominal cavity (*echinorrhynchus*, mites, etc.), operations (castration), etc. The symptoms are prostration, fever, loss of appetite and groaning when the stomach is pressed, or during evacuation.

**Diagnosis.**—As will be seen from the foregoing, diagnosis of acute peritonitis is very difficult, especially in the larger animals. But if the existence of pain in the abdomen, with accumulation of exudations therein, can be proved, then diagnosis is certain. The latter symptom only can protect one against confusing it with enteritis and colic. Another characteristic sign is the very small wiry pulse. Examination of breathing and pulse are of little value in preventing the disease being mistaken for pneumonia, pleuritis or endocarditis. With regard to confusion of the peritonitic exudation with other conditions, compare the paragraph on the Differential Diagnosis of Ascites.

**Therapeutics.**—The treatment is partly local, partly internal. The inflammation of the peritoneum may be locally combatted by external application of cold bandages and, in smaller animals, by laying ice-bags on the stomach. The custom of rubbing in mercurial ointments cannot be universally recommended in domestic animals, owing to the danger of salivation; It can be used only on horses and dogs. On the other hand, turpentine and mustard oil may be rubbed in on ruminants. When the exuded matter accumulates excessively, its removal may be attempted by puncture. In ulcerative peritonitis, a wide opening of the abdominal cavity by laparotomy is often the only method of saving life. In such cases the cavity must be afterwards carefully disinfected and washed out with a weak solution of salicylic and boracic acids. Injections of Lugol's iodine solution are also made for the same purpose. Internal

treatment consisted formerly in administration of remedies to allay inflammation and of strong purgatives (tartar emetic, saltpetre, Glauber's salt). As, however, these remedies stimulate and increase peristalsis, practitioners are cautioned against their use, because, by spreading the peritonitic exudations over a wider surface, they also spread the irritation. We should rather prescribe calomel as a disinfectant for the bowels. In later years opium has been given to reduce the peristaltic action of the bowels, and this in large doses (horse 2½ drams, ox 4 drams, dog 1½ to 7½ grains of the powder). The increased constipation thus produced may be combatted by simultaneous lukewarm clysters and infusions. Where much fluid collects in the abdominal cavity, diuretics may be used (digitalis, squills, strophanthus, caffeine, juniper berries, oil of turpentine and acetate of potash), or such as stimulate the flow of saliva (pilocarpine, arecoline). In so far as the primary conditions which produced the peritonitis can be attacked, these must naturally be first of all dealt with (disinfection of the uterus in metritis, of the bladder, in cystitis; herniotomy in strangulated hernia, opening of abscesses, etc.). When the debility is very alarming, or in order to combat fever, subcutaneous injections of camphor are suitable remedies.

## CHRONIC PERITONITIS.

**Etiology.**—A diffuse chronic peritonitis is much rarer than the acute form, and occurs most frequently in cattle. It may develop from the acute form, or arise independently. Of the latter kind the causes are rather obscure. In cattle one has to consider the passage of foreign bodies from the second stomach towards the abdominal cavity; in dogs the punctures which are often made to relieve ascites; and in all animals chronic inflammatory processes in the organs covered by the peritoneum (womb, kidneys, liver). Finally, there are chronic tuberculous, sarcomatous and carcinomatous forms of peritonitis as part phenomena of tuberculosis (cattle and pigs), sarcomatosis (horses) and of generalised cancer.

**Post-mortem Lesions.**—The anatomical changes in chronic peritonitis are confined very often to limited areas (perihepatitis, perimetritis), and consist chiefly in a gradually increasing inflammatory proliferation of the peritoneal connective tissue, which often leads to very considerable thickening

of the peritoneum, and to the growth of regular capsules of connective tissue around certain organs, and, at times indeed, to local fibrous, cartilaginous, or even bony areas. At the same time the organs grow fast to each other, and to the peritoneum, thereby producing stretching, compression and shrinkage of them. In this deforming or distorting peritonitis, which may be best observed in pigs, the bowel-loops grow together into knotted convolutions, whose interstices are often filled with a purulent, gelatinous, oily exudation, which later becomes caseous or chalky. Chronic peritonitis with abundant fluid exudation is rare; but it occurs, for instance, in the sarcomatous peritonitis of horses.

**Symptoms.**—These are generally the same as in acute peritonitis, only much less severe. The disease is consequently also more difficult to demonstrate, unless it follows the acute form. Its course is very slow. In *cattle* the signs of colic and of pain on palpation are generally lacking, and often the only symptom left is the daily increase of circumference in the abdomen, and this in spite of lost appetite and continuous diarrhoea. To this an edematous swelling of the lower abdominal region may be added later. The same signs are sometimes noticed in *horses*, in whom, besides the symptoms just named, intermittent, but only brief, attacks of colic may be noted, along with great sensitiveness in the flanks, accompanied by a rapid but small pulse and medium fever. In *dogs* the differentiation between this disease and ascites is very difficult and often quite impossible.

**Therapeutics.**—In many points, the treatment corresponds with that of ascites. In both complaints, when there is a considerable quantity of exuded matter, diuretic and laxative remedies are to be used along with puncture. In the beginning, and with dryer exudation, we recommend warm wraps in the form of Priessnitz's bandages, for the abdomen, with internal administration of iodine salts (iodide of potassium and of iron). Rinsing the abdominal cavity after puncture is also to be recommended with smaller animals. For this we generally use Lugol's solution diluted with sterilised water.

#### ABDOMINAL DROPSY. ASCITES.

**General Remarks.**—Ascites (dropsy of the abdomen) is an accumulation in the abdominal cavity of a non-inflammatory

exudate, and occurs in all our domestic animals, including poultry. In a pronounced form it is most frequent in dogs, especially in very young or very old ones. In the Berlin dog-hospital, 318 cases of ascites were met among 70,000 dogs treated, or 0.5 per cent. of the whole. It is also not uncommon in sheep, cattle and goats. It is certainly rare among horses, and Woodger says that in 22 years he only saw it once. The reason probably lies in the greater activity and more regular feeding hours of horses.

**Etiology and Pathogenesis.**—Ascites is no uniform or independent disease, but rather a symptom of various other primary disorders. In this sense we may indicate its causes as follows :—

i. *Chronic affections of the heart, lungs, kidneys, portal veins or mesenteric glands.* These have partly, general disturbances of circulation as a consequence (heart, lungs, kidneys); and partly, limited disturbances (liver, portal vein, mesenteric glands); they cause venous engorgement and transudation of lymph.

(a) Among *heart-diseases* we must mention : old-standing and not sufficiently compensated valvular defects; chronic pericarditis and epicarditis with enormous serous exudation, or adhesion of the pericardium to the heart (*cor villosum*); and hydropericardium.

(b) Of *lung-diseases* : Emphysema of the lungs, interstitial processes with consecutive sclerosis and atrophy (tuberculosis).

(c) Of *kidney-diseases* : Interstitial nephritis (*Bright's disease*); and degenerative process.

(d) Of *liver-diseases* : Above all interstitial hepatitis, which leads on to cirrhosis of the liver (*e.g.* in distomatosis, hydatid disease); adenoma of the liver, etc.

(e) Among *diseases of the portal vein* : Compression by tumours, thrombosis, etc.

(f) Of *diseases of the mesenteric glands* : Caseous degeneration and atrophy, as, for instance, in the enteritis of foals.

In all the above-named diseases the flow of blood from capillaries and veins (*i.e.* the flow of lymph) is hindered; engorgement, with increase of intravascular pressure, occurs, complicated with increased permeability of the vascular walls, all caused by disturbed nutrition of the vascular endothelium, resulting from engorgement. These are conditions which easily

explain the extravasation of fluid-blood from the vessels. In cases of engorgement in the region of the portal vein and its branches, the capillaries and veins of the peritoneum, which form the commencement of that system, are especially affected.

2. *Hydramia*, that is increase of the watery constituents of the blood, and a lessening of its solid contents, especially albumen ;—the so-called *hydramic or cachectic ascites*. This hydramia may appear either as a primary or a secondary condition, after severe, prolonged and very wasting sickness, thus especially in sheep and cattle (so-called dry rot, *cachexia aquosa* ; worm-diseases, *cachexia verminosa*, etc.). Here the transudation is consequent upon an alteration in the walls of the vessels. Along with ascites, dropsy of the breast, pericardium and skin are also present.

3. *Carcinomatous, sarcomatous and tuberculous new growths of the Peritoneum*, as well as healed inflammations of the same, are also considered to be causes of ascites. But it would here be a question of exudative, rather than transudative processes, that is to say, of chronic peritonitis, although the occurrence of true abdominal dropsy on the basis of above-named changes cannot be denied. But one must not forget that ascites and chronic peritonitis are by no means easy to differentiate. In advanced stages of tuberculosis in old cows, we believe we have frequently discovered ascites caused by tubercular nodules, seeing that there were no inflammatory changes of the peritoneum. On the other hand, we failed to find any trace of ascites in a dog ten years old, which had carcinomata spread over almost all its organs, and whose peritoneum was particularly severely affected.

**Post-mortem Lesions.**—The fluid collected in the abdominal cavity of a horse may exceed 33 gallons in quantity. Brusasco, for example, drew 38 gallons from a horse in three hours. From a dog Hordl drew 3½ gallons. This exuded liquid closely resembles blood-serum, and is sometimes clear and pale yellow, at others, opalescent and slightly turbid and greenish-yellow, owing to suspended fatty-degenerated epithelium, or again it is tinted red by red blood-corpuscles (especially in dogs), and occasionally charged with fibrin-flakes. The amount of albumen is usually small ; in a dog we found it to be 3.5 per cent. The specific weight is correspondingly light (in men 1012, and in a horse we also found 1012). The peritoneum is often merely saturated with moisture, but in prolonged cases of ascites

it becomes whitish, opaque and thickened, as a result of swelling, fatty degeneration and desquamation of its epithelial cells, and of stimulated new growth. Besides this, a formative irritation of the tissue takes place (Ziegler), whereby moderate small-celled infiltration of the connective tissue is produced and, in cases of long duration, even new growths of connective tissue, with considerable thickening of the serosa and partial adhesion of the peritoneal layers. Where large accumulations exist for a long period, the various organs of the abdominal cavity appear pale, anaemic, and even atrophied. The viscera are often contracted, the diaphragm is relaxed and thrust very much forward, whereby the thoracic cavity is narrowed, and the lungs are more or less affected. Finally, other changes are found, some of which are the cause of the ascites, others its consequences, viz., changes in the heart, lungs, liver, etc. anaemia, hydramia and emaciation.

**Ascites chylosus.**—This occurs after rupture of the larger lacteal vessels and admixture of chyle with the exuded matter, which consequently assumes a milky character. Gray describes a case in a cat.

**Symptoms.**—In very severe ascites these consist in:—

1. A gradual *enlargement of circumference* of the abdomen which occasionally, however, develops quickly. This is accompanied by sinking of the flanks and back. In smaller animals the neighbourhood of the navel is strongly arched; and it is usually the abdominal wall in dogs, sheep (especially shorn ones), cattle and cats which shows this bulging out, and most conspicuously just behind the ribs. The joints of the cerebral column show very distinctly and, combined with the depressed back, sunken flanks and pendulous belly, present an aspect which is truly characteristic of this disease.

2. *Palpation* of the upper part of the abdominal wall, which is slack, and of the lower part, which is very tense, produces the feeling of *fluctuation*. Thus, if one lay the hand on one side of the abdomen, and then apply pressure to the other side in short, quick pushes, splashing or wave-like movements can be felt. The same can be perceived in the larger animals on palpation through the rectum, that is, if there be a large accumulation of fluid. On applying the ear to the animal's side, the sound of splashing liquid can be clearly heard. In smaller animals the sound may be produced by merely shaking them. But in both cases care must be taken not to

confound abundant fluid contents of stomach or intestines with ascites.

3. *Percussion* in the region of this accumulation produces a *dull (empty) sound*, provided that on the thinner layers percussion be not too strong, nor the fluid displaced by pressing in the pleximeter too deeply. This dull sound occurs in the lower portions of the abdominal wall, its upward boundary being clearly defined horizontally, where it changes to a tympanitic bowel-tone. Of great importance is the change of this dull percussion-note according to the animal's position. If we lift a small animal by its fore feet and set it on its hind quarters, we are at once struck by the extraordinary bulging of the abdomen directly over the symphysis, and by the percussion-note of this part, which note again is limited by a horizontal line corresponding to the surface of the fluid. The contrary occurs if the animal's position be reversed. The fluid then collects upon the diaphragm, and the dull sound begins at the cartilage of the sternum. As respiration is hindered by pressure of the fluid on the diaphragm, and even becomes at times dyspnoëic, the animals struggle to escape from this position.

4. *Breathing* is quickened and impeded by the forward thrusting of the diaphragm and the lessened power of contraction in the abdomen, and is performed with marked co-operation of the accessory muscles, particularly of those which move the ribs. In severer cases it is dyspnoëic, pumping, and accompanied in horses with much action of the nostrils.

5. *There is no fever*, unless produced by complication of the primary disease. The *pulse* is usually accelerated, grows steadily smaller and is at last thready. The heart-beat is easily excited, and later becomes palpitating. The *mucous membranes* are pale; the conjunctiva in sheep is infiltrated with fluid and the skin also very white. The *coat* is lustreless and ruffled, and the extremities feel cold. Dogs often adopt a sitting posture with the fore feet widely stretched, and are very unwilling to move, are soon tired, and only lie down continuously when very exhausted, whereby decubitus is often developed, as well as involuntary urination, consequent on spasm of the sphincter vesicæ.

6. In later stages *appetite gradually diminishes*, and also rumination and movement of the paunch. In dogs the appetite seems for a long time unaffected, but becomes fastidious, and occasionally they vomit. Defecation is alternately retarded

and diarrhoeic; sometimes tympany supervenes; urination, at first increased, later becomes diminished. The secretion of milk gradually diminishes, and by degrees the signs of general chronic disturbance of nutrition manifest themselves.

**Course.**—This is chronic, lasting months and even years. The bodily wasting continues, signs of anasarca appear on the hinder parts, viz., on the prepuce, udder, hypogastrium and thighs, partly caused by pressure of the transuded matter upon the veins, partly by general and gradually developing dropsy. The animals grow ever weaker and more wretched, till at last they cannot rise up, the eyes sink deep into their sockets, dyspnoea and decubitus obtain the mastery, profuse diarrhoea prevails (in cows sometimes retching and vomiting), and death finally occurs by paralysis of the heart or lungs or by general marasmus.

**Diagnosis and Differential Diagnosis.**—The diagnosis of ascites is made by inspection, palpation, percussion and auscultation of the abdomen. To ensure certainty, an exploratory puncture may be made in the case of the dog. The latter confirms the diagnosis even in cases of smaller accumulations of liquid, in which otherwise an opinion is always very hard to form, and with the larger animals almost impossible. Indeed, ascites is more easily recognised in the smaller animals, because examination can be more thorough.

But more difficult than the demonstration of the presence of ascites is the discovery of its exciting cause, although, both for therapeutics and prognosis, this is of first importance. General dropsy may be most easily established as the cause of ascites, if there be a simultaneous dropsy of the skin (anasarca) and subcutaneous cellular tissue, as well as diffuse dropsy of the cavities. It is more difficult to recognise specific heart or lung affections (see the respective chapters), and particularly interstitial chronic hepatitis, which is a frequent source of ascites, but difficult to diagnose, owing to the fact that shrinkage of the liver and other characteristic signs, such as icterus, cannot always be detected. Enlargement of the liver, accompanied by prominent changes of its surface, can be more certainly recognised by palpation as the cause of abdominal dropsy. The presence of biliary pigment in the urine is a symptom of no importance, as it often occurs in dogs with the slightest gastro-intestinal catarrh, and in severe degeneration of the

liver (e.g., from liver-flukes) such pigment is altogether absent. In dogs the search for an exciting cause is often rendered more difficult by a combination of two ailments, for instance, of a valvular defect with cirrhosis of the liver. Finally, new growths of the peritoneum may, in rare cases, be positively demonstrated by palpation.

Of diseases which may be confounded with ascites we have to consider :—

1. *Peritonitis*.—This is accompanied by fever, great disturbance of general health, signs of pain, sensibility to palpation. Moreover, the specific weight of the peritonitic fluid, as obtained by puncture, is higher.

2. *Pregnancy*.—May be easily tested by manual investigation. The animal's general health is undisturbed, and the belly distended more on one side than the other.

3. *Obesity*.—Especially in older dogs, if combined with some valvular defect and much shortness of breath, this may lead to mistake. Differentiation must then be based upon auscultation and experimental puncture (inspection and palpation are not sure guides).

4. *Hydrometra, pyometra, dropsy of the membranes of the fetus* (especially in cows), *cystic degeneration of the ovaries*, *abdominal cysts*, especially *abnormally large tumours* in the abdominal cavity (Cunningham found in a two-year-old foal a new growth weighing 236 lbs.) and *cystic kidneys*. All these may be recognised, partly by examination through the rectum or vagina, partly by exploratory-puncture, partly by the stability of the various regions as proved by palpation and percussion. Whereas in ascites the results of percussion vary with the animal's position, with abdominal or ovarian cysts they remain constant. An abnormal accumulation of dung in the rectum may also lead to error.

5. *Paralysis of the bladder with excessive distension*. Here palpation reveals a spherical, fluctuating body in the region of the bladder; and percussion, that its position does not change. Disturbed urination, use of the catheter and an exploratory puncture may all assist in diagnosis.

6. *Rupture of the urinary bladder*, with accumulation of urine in the abdominal cavity. This occurs most frequently in oxen after lodgment of urinary calculi in the urethra, and may be diagnosed from the precedent colic, cessation of urinary discharge, empty bladder, urinous perspiration, fever and appearances of collapse.

**Prognosis.**—This is so far unfavourable, as that those anatomical changes in the organs which are the true causes of ascites cannot, as a rule, be removed, and that alterations in the blood (hydræmia) have already taken place before the veterinary surgeon is called in. From our own experience we consider that the prognosis is relatively most favourable in the case of young dogs, and in a few such we have seen the disease spontaneously disappear. In older animals only a temporary recovery can be expected, seldom a permanent cure. The prognosis of chronic peritonitis, with which this complaint was often confounded, especially in horses, is more favourable.

**Treatment.**—This should first of all aim at removing the affections of heart, lungs or liver which lie at the root of the ascites. As these are mostly beyond the reach of medical art, the treatment must fall back on regulation of diet (albuminous food) and removal of the accumulated liquid. For these ends, administration of diuretics and purgatives is most usual; also sialagogues and diaphoretics should be tried. Of diuretic remedies we recommend digitalis in combination with juniper berries and acetate of potash, squills, diuretin and caffein are also good diuretics. Tincture of strophanthus and calomel are prescribed occasionally. Drastic remedies are inferior to diuretics, for, when used continuously, they cause weakness. For dogs, gamboge, croton oil and jalap have been mostly used. For all domestic animals, pilocarpine and arecoline should be especially used to stimulate salivation (sialagogues). They must be given in small doses—and very cautiously where there is heart affection:—viz., pilocarpine to horses subcutaneously, 3 grains; cattle,  $4\frac{1}{2}$  grains; dogs,  $\frac{1}{8}$  th grain; and arecolin to horses, 1 grain. The former practice of dosing dogs with sublimate is to be condemned, although it occasionally met with success.

Finally, with regard to puncture for ascites, it is advisable, and indeed absolutely necessary, in all those cases in which the exuded matter by its mass and weight presses the diaphragm too far forward, thus producing dyspnoea and imminent danger of suffocation. In all other cases the operation is useless, for as stoppage still remains, the abdomen quickly fills up again, and repeated tapping of this accumulation draws off too much albumen from the animal. We must also point out that with older dogs the apparently safe operation of puncture may sometimes cause their sudden death, when, for instance,

the animals grow very excited and offer resistance (heart-shock). Also, if the ascitic fluid be allowed to run off too quickly, insensibility and paralysis of the brain may result. The best results may be expected from puncture in young dogs; and we have often noticed a cure from an introduction of the trochar. The injection of Lugol's iodine solution into the abdominal cavity after puncture, and which is recommended by many, is probably only of benefit in cases of chronic peritonitis and of ascites following on degeneration of the peritoneum.

## CHAPTER VI.

### DISEASES OF THE SPLEEN.

**General Remarks.**—Although, as will appear from the foregoing chapters, diseases of the spleen are by no means rare, they have in general but little interest for special pathology and therapeutics. For, with very few exceptions, they cannot be diagnosed during life, and are therefore only subjects for pathological anatomy, to the text-books on which we recommend our readers to turn. Moreover the pathological changes of the spleen are mostly secondary processes of infectious disease, anomalies in the blood and other ailments, which are elsewhere treated (pyæmia, anthrax, leucæmia). Finally, a great proportion of spleen-diseases belong to the sphere of surgery (traumatic affections of the spleen). But in order not to lack completeness, and to show how small is the practical importance of these complaints—they are not even mentioned in many clinical text-books on human medicine—we will deal briefly with the following three most common diseases of the spleen:—

1. Hyperæmia of the spleen.
2. Inflammation of the spleen.
3. Rupture of the spleen.

**Hyperæmia of the Spleen.**—Active or congestive hyperæmia of the spleen occurs secondarily in most infectious general diseases, especially in all cases of septicæmia (anthrax, sepsis, pyæmia, the erysipelas of pigs, etc.). Primary congestion of the spleen is thought to arise from overloading the stomach, or from a too sudden change from meagre diet to full rations (acute plethora). Acute hyperæmia of the spleen may lead to rupture, inflammation and hyperplasia of the organ. It is clinically marked by enlargement and swelling, but the latter cannot usually be confirmed by palpation and percussion, so that an acute congestion of the spleen cannot, as a rule, be diagnosed, but only suspected from the accompanying general disorder. A *passive* hyperæmia of the spleen (*chronic congestion of the spleen*) arises in all disturbances of circulation which hinder

the discharge of the splenic-vein, also in all chronic affections of the liver, heart and lungs. If long persistent, it leads to induration of the connective tissue.

**Inflammation of the Spleen.**—Most frequently results from traumatic injuries of the organ, or wounds inflicted from without or from within, pressure, blows, crushing, shock, etc. This inflammation is also an accompanying sign of pyæmia, and may, moreover, develop from an acute tumour of the spleen. We distinguish anatomically, a simple *Splenitis*, known by extreme swelling and enlargement of the spleen, grey colour of the pulp and cellular infiltration of the latter, and *Abscess*, or purulent inflammation of the spleen, which is usually of traumatic or pyæmic (metastatic) origin, and may appear either singly or in numbers. The localised abscesses of the spleen are either encapsulated, or break through into the abdominal cavity. Finally, a third form is identified as *Perisplenitis*, an inflammation of the serous covering of the spleen, which leads to thickening of the capsule and adhesion of the spleen to its adjacent organs.

The *Symptoms* of inflammation of the spleen are of very undecided nature. In most cases the ailment cannot therefore be demonstrated during life. This is the more easily explicable because it has been found that very serious inflammatory changes in the spleen seem to produce no visible change in an animal's general condition. Thus Möbius found on slaughtering a cow, which was apparently thoroughly sound and well nourished, that its spleen was changed into an abcess holding 5½ pints of pus. If signs of illness be manifested, these are mostly of uncertain character, consisting in gastric disturbance, great emaciation, colic and fever. An inflammation of the spleen can then only be with certainty diagnosed, when enlargement of the organ and painfulness in that region can be proved by inspection, palpation or percussion. According to Notz, traumatic, haemorrhagic swelling of the spleen occurs in sucking calves when they have been trodden on or kicked by their mothers. They suddenly grow very ill, refuse all food and lie with half-shut, weeping eyes. Their pulse and breathing are accelerated, peristaltic action is suppressed, the abdomen is moderately full, and the ears cool. Death follows in from 12 to 18 hours. Traumatic splenitis is relatively the easiest to recognise. The *treatment* of this form of inflammation is purely symptomatic.

**Rupture of the Spleen.**—This is generally of traumatic origin, but may occur as a consequence of congestion of the spleen or of splenitis. The pathological changes vary according to the position and size of the rupture. If superficial and small, and the capsule uninjured, a small haematoma arises in the form of a boil, which may heal up by resorption and cicatrisation. In the same way, rents in the capsule may also heal, as has sometimes been noticed in horses on subsequent dissection. More serious haematomata of the spleen show their presence by signs of colic, lassitude, paleness of the mucous membrane and steady wasting. But they can only rarely be proved by palpation and percussion. In extensive ruptures of the parenchyma of the spleen, e.g., in great rents of the capsule, the animals reveal signs of internal haemorrhage, anaemia of the mucous membranes, cold extremities, rapid failure of strength, fear, sweatings, imperceptible pulse, fall of bodily temperature and death in a few minutes or hours. Sometimes periodically recurrent haemorrhage is noted, with alternate improvement and deterioration of general health. The treatment consists in administration of styptic remedies (ergot, tannin, sugar of lead and preparations of iron).

**Diseases of the Pancreas.**—These have still less clinical interest than affections of the spleen. Mégnin and Nocard have described a case of pancreatitis in a horse. It displayed slight weakness, weakness, lessened appetite, constipation, emaciation and icterus. Postmortem revealed a very great enlargement of the pancreatic duct, which was occluded by a plug of albumen, compression of the common biliary ducts by the enlarged pancreatic duct, induration of the tissues of the pancreas, along with catarrh of the salivary ducts. New growths in the pancreas, which occur especially in dogs in the shape of adenomata and carcinomata, have also more anatomical than clinical interest. A few cases have been described by Nocard and Friedberger. In cattle, salivary concretions occur in the pancreas similar to those in parotitis. The pancreatic duct and its larger ramifications are dilated like a string of beads, their walls thickened, and the expanded ducts filled with small whitish stones, partly angular and faceted or cylindrical, varying in size from a millet-seed to a hazel-nut, and consisting chiefly of carbonate of lime. Among the cattle of Japan *distomum pancreaticum* occurs very often in the *ductus Wirzburgianus* (Janson).

**Parasites in the Abdominal Cavity.**—1. In horses are found: *filaria papillosa*, *strongylus armatus* and *cysticercus fistularis*. 2. In ruminants, *cysticercus tenuicollis*. 3. In pigs, *cysticercus cellulosae* and *tenuicollis*, *sclerostoma pinguicola*. 4. In carnivora, *echinococcus-cysts*. 5. In rabbits, *cysticercus pisiformis*.

## CHAPTER VII

### DISEASES OF THE URINARY ORGANS.

#### INFLAMMATION OF THE KIDNEYS. NEPHRITIS.

**Classification.**—Nephritis has been at different times and on various grounds divided into a great many forms, based upon anatomical or etiological reasons, or named according to their origin or chief symptoms. Without wasting time upon this multitudinous subdivision, we select the following clinical forms for consideration, as being most in agreement with the present state of veterinary pathology.

1. *Acute nephritis*, formerly known as "acute Bright's disease." This may be of infectious, toxic, rheumatic or traumatic nature, and may occur as glomerulo-nephritis, or as acute diffuse nephritis.

2. *Chronic nephritis*, formerly called "chronic Bright's disease." Included under this head, are chronic parenchymatous and chronic interstitial nephritis, as well as the final stage of the latter, the so-called granular and atrophied kidney.

3. *Suppurative nephritis* following on suppurative metastasis.

4. *Pyelo-nephritis*, viz: inflammation of the kidneys and pelvis of the kidney, which often occurs in cattle.

**Chief Clinical Forms of Nephritis in Man.**—In addition to suppurative and pyelo-nephritis, we also clinically distinguish three chief forms of inflammation of the kidneys, viz:—

1. *Acute nephritis*, usually developed in connection with infectious disease, and ending mostly, either in recovery or death, but seldom passing into the chronic interstitial form. Chief symptoms: oliguria, excessive albuminuria, great specific gravity of urine, hyaline casts, epithelia of the kidneys, white, and occasionally also red, blood-corpuscles in the urine.

2. *Chronic parenchymatous nephritis*, generally fatal, or leading to atrophy. Course is slow. Chief symptoms: moderate oliguria, intense

albuminuria, higher specific gravity, casts, white blood-corpuscles, fatty-degenerated epithelia and waste substance; red blood-corpuscles only rarely; dropsey. *Anatomically characterised by kidney-enlargement (large, white kidney) and intense desquamation of the epithelium.*

3. *Granular and atrophied kidney, induration or sclerosis of the kidney.* Develops slowly, sometimes from the second form. Chief symptoms: Polyuria, slight albuminuria, very low specific gravity, scanty formative elements in the urine. No dropsey, but, on the other hand, hypertrophy of the heart. *Anatomically characterised by shrinking of the kidneys and proliferation of connective tissue.*

**Definition of Bright's Disease.**—Under this name some authorities include all kidney diseases which are accompanied by albuminuria, and others restrict the term to true inflammation of the kidneys, including amyloid kidney; others again to primary interstitial nephritis. In view of such a divergence of opinions, it would be well to reject this old name of "Bright's Disease," and to set the anatomical nomenclature in its place. In veterinary pathology it has caused great confusion, especially in cases of haemoglobinuria of horses.

#### I.—ACUTE INFLAMMATION OF THE KIDNEYS. NEPHRITIS ACUTA.

(*Parenchymatous, catarrhal and hemorrhagic nephritis, glomerulonephritis, inflammatory œdema of the kidneys, opaque swelling of the kidneys.*)

**Etiology.**—Acute nephritis occurs in all domestic animals from various causes, and is commonest in horses, cattle and dogs. First, it may develop from a simple hyperæmia of the kidneys. It may result from traumatic causes, blows on the back, falls, over-exertion when pulling or too sudden reining-in when going at great speed. Not seldom it is produced by chill, especially in horses. It is necessary to bear in mind the close connection between the skin and the kidneys, and the indirect action of any suppression of the activity of the skin. Further, acute nephritis very often occurs during infectious diseases. These almost all cause an infectious or toxic nephritis by reason of the excretion of bacterial poisons through the kidneys. In lighter cases this shows itself rather as an opaque swelling and fatty degeneration of the epithelium of the kidney. Here we have most frequently to consider septic and pyæmic processes, such as puerperal septicæmia in cattle. But other infectious complaints, such as influenza, murrain, strangies, angina, erysipelas, anthrax, foot-and-mouth disease, distemper, bronchitis, etc., are also sometimes complicated with acute nephritis.

With regard to glanderous and tuberculous nephritis, see the respective primary diseases concerned.

The pathology is the same in cases of nephritis produced by poisoning. For instance, those caused by meadow-saffron, cantharides, oil of turpentine, croton oil, male-fern, squills, etc., and above all, many diseases in cattle which are termed "haematuria." Tar, carbolic acid, iodoform, and especially the metallic poisons, such as phosphorus, arsenic, lead, mercury, etc., all have the same effect upon the kidneys. Also the fungi of mould, smut, blight or ergot (*hyphomycetes*, *uridines* and *ustilagineæ*) excite inflammation in the kidneys, as well as certain insects, e.g., caterpillars and plant-lice on cabbages. Even too liberal a feed of raw potatoes and certain kinds of cotton-seed meal, will do the same. Secondary nephritis may arise from the spread of cystitis and pyelitis. Finally, of great practical importance is the nephritis which arises in the course of haemoglobinaemia of horses, which is caused by haemoglobinous congestion of the kidneys. It was formerly held to be the primary cause of the complaint just named.

**Pathological Anatomy.**—Acute nephritis occurs, according to Kitt, in various forms in our domestic animals.

1. *Nephritis parenchymatosa* is a joint designation for all those cases in which rough anatomical methods cannot decide whether we are dealing with an opaque swelling or with desquamation, necrosis of the epithelium, serous or sero-fibrinous exudation. In such instances the kidneys are not swollen, or only very slightly, more easily detachable from the capsule and, on dissection, of a dull, washed-out colour, that is to say, grey or greyish-red, or spotted with yellowish brown. Their medullary substance is more or less hyperæmic, even stained dark red, the Malpighian corpuscles stand out prominently, while the body of the kidneys is of normal or softened consistence. Under the microscope one finds the capillaries and veins enlarged and fully charged with blood, there are fibrinous and granular exudations between the tubuli uriniferi, granular, hyaline and epithelial casts (cylinders), detached epithelial cells, fatty degeneration, opacity, swelling and formation of vacua.

2. *Nephritis parenchymatosa hemorrhagica* is characterised, in addition to the signs already detailed, by many red, minute blood-points, which give the kidney an almost black-red external appearance, which on section is dull grey, spotted with red, and in its medullary substance quite a deep red (*erysipelas of pigs*).

3. *Nephritis acuta diffusa* is distinguished by excessive enlargement and increase of weight (twice or thrice the normal size), as well as by the soft, friable nature of the tissue. The capsule is very loose, the upper surface shows a very bloody and bright colour, with grey and yellowish-grey patches. The microscope reveals very considerable enlargement of the capillaries with extravasation of blood into the tissue-clefts, leucocyte casts in the distended urinary ducts, as well as cellular infiltration between them. The glomeruli are often very full of blood and covered with sanguinary extravasations.

**Symptoms.**—The most important, and sometimes the only symptoms, are in connection with the urine (e.g., in the course of severe infectious disease). This is first exceedingly rich in albumen. It is also much reduced in quantity, and in graver cases the discharge of urine is for several days together quite suspended. Thus Funk noticed anuria in a cow lasting for five days, and Friedberger a similar case in a horse which lasted seven days. The causes of this are stoppage of the urinary canals by exuded cylinders (casts), extreme inflammatory swelling of the tissue of the kidneys and consequent compression of the ducts, as well as a diminished secretion of water by the affected organs. The specific weight of the urine is always much increased. The liquid is itself of thicker consistency, often slimy, turbid and discoloured. Sometimes this develops to haematuria.

The microscope reveals the various kinds of urinary casts in great quantity, some covered with cells and white and red blood-corpuscles, and further, according to the desquamative, interstitial or haemorrhagic character of the complaint, one finds numerous epithelial cells and white or red blood-corpuscles. The discharge of urine often occurs only in drops and with much pain and pressure (dysuria, strangury). On exploration through the rectum, the bladder is always found empty. The region of the kidneys (loins) is painful under palpation, and the in-drawing of the back provoked by pressure upon the kidneys or lumbar region is very painful for the animal. The kidneys are found on examination (when possible) to be enlarged and sensitive to touch. Frequently, and especially at the commencement of the disease, intense paroxysms of pain, so-called renal colic, are manifested. The animal's back is stiffly bent, and its gait is strained and staggering. It rises with difficulty, and remains standing, usually with straddled legs or feet gathered

under its body. Sometimes also in walking the foot on the ailing side is dragged somewhat, or not advanced so far as the other. In male animals one testicle is often drawn up.

Appetite is suppressed from the first; and in dogs vomiting is sometimes noticed. Constipation then generally sets in, to alternate later with diarrhoea. Bodily temperature is slightly or moderately raised, i.e., so long as there are no uræmic attacks. The pulse, which at first is strong and hard, grows afterwards more rapid but weaker. Nothing is as yet known of hypertrophy of the heart occurring in domestic animals after an acute nephritis lasting several weeks; also the affections of the lungs noticed in men during the later course of this disease (bronchitis, pneumonia) have not yet been noticed. On the other hand, hydramic swelling of the subcutis develops in the domestic animals, viz., on the head, breast, belly, scrotum, joints and in the abdomen, as well as dropsy in the cavities of the body.

In severer cases of nephritis with fatal ending the symptoms of uræmia occur in addition to the above, viz., stupefaction, spasms and dyspnoea. Pflug found, during the final stages in a cow with bilateral nephritis, the most violent eclamptic attacks, with opisthotonus and (in the intervals) complete coma. We ourselves observed in a horse with bilateral acute diffuse nephritis, about twelve hours before death, considerable stupor, reeling, staggering, general sweating, vague muscular twitchings over the whole body, combined with extraordinary increase of bodily temperature (almost 108° F.).

**Uræmia.**—Except in the dog, cases of true uræmia are not common among our domestic animals. Dammann once saw in a sheep epileptiform spasms alternate with sopor. In dogs, vomiting, weakness resembling that of paralysis, coma and fall of bodily temperature are noted as uræmic symptoms connected with urinary calculi, stricture of the urethra and paralysis of the bladder. In a horse we observed pronounced epileptiform attacks. In regard to the nature of uræmia we must refer our readers to the text-books of general pathology. It consists, in brief, of an accumulation of urinary constituents in the blood and tissues, first, the products of metabolism resulting from decomposition of its albumen (ptomaines, leucomaines and toxines), and then the urea and salts of potash. Traube's theory, according to which uræmia is essentially the result of an acute oedema of the brain, and also that of Frerich, which states the conversion of the urea into ammonium carbonate in the blood to be the true cause of uræmia, have neither of them found confirmation. The symptoms of uræmia in man are head-ache, somnolence, restlessness, delirium, nausea, vomiting, a feeling of anxiety, slight twitchings, as well as severe eclamptic fits, after which uræmic amaurosis or deafness frequently remains. Later on follow vomiting, sobbing, diarrhoea, excretion of urea on the skin producing itching, retardation of the pulse followed by

quickening, a bodily temperature either abnormally high or abnormally low and dyspnoea

**Course and Prognosis.**—Acute inflammation of the kidneys is usually decided within eight to fourteen days, but death may ensue in very few days. As a precautionary measure it is better to regard the prognosis as unfavourable, as the cases of recovery and death are about equal. But the appearance of the uræmic symptoms is very unfavourable. Death usually results from œdema of the lungs or brain. Development into chronic nephritis is rare.

**Differential Diagnosis.**—Acute nephritis is often mistaken for an inflammatory affection of some other abdominal organ, thus for peritonitis, colic, enteritis, cystitis or metritis, and is consequently overlooked. But this can only occur when we omit to examine the urine, which will in all cases ensure a correct diagnosis of nephritis without much difficulty. Moreover, a careful local investigation of bladder, uterus, etc., should prevent all mistake.

**Therapeutics.**—The treatment of acute nephritis consists first in rest and avoidance of all irritating food, as well as of irritant medicines, such as turpentine or cantharides. For dogs, as for men, an exclusive milk diet is to be recommended. Then one must endeavour to relieve the kidneys by diaphoretics, laxatives, diuretics, and perhaps also by local derivatives in the region of the kidneys, of which the application of cold or moist or warm packs would be most suitable. Perspiration can be produced in the larger animals by diligent rubbing with straw, wrapping in warm clothes, use of moist, warm (Priessnitz's) bandages about the rump; and a flow of saliva may be stimulated by subcutaneous injection of pilocarpine (2 grains for horses; 7 grains for cattle;  $\frac{1}{2}$  to  $\frac{1}{4}$  grain for dogs), as well as arecoline ( $\frac{1}{2}$  grain for a horse). Smaller animals, such as dogs or pigs, may be made to perspire by hot baths, the temperature of which is gradually raised to that of the body, or by hot cloths. Of purgatives the most drastic are the best, because of their intense power of driving out water (calomel, aloes). But such as excite the kidneys (croton oil) must be avoided. The same applies to diuretics, of which the alkalis, particularly acetate of potash, should be used, and, if threatened by heart-failure, digitalis, but especially diuretin and caffeln. Against eclamptic fits, bromide of potash, chloral hydrate and inhalation of chloro-

form must be tried. As regards the former common custom of administering astringent, styptic remedies, such as tannin, sugar of lead or sulphate of iron, one may always expect them to produce vascular contraction, and, therefore, to reduce inflammation and act somewhat as antiseptics. We have hitherto used tannin in the form of China bark (cinchona)—also in the treatment of fever—as well as bearberry, and, from the results so far obtained, see no reason why we should not further recommend its use.

**Hyperæmia of the Kidneys.**—This is either a passing condition or altogether secondary, which consequently has rarely to be diagnosed as an independent ailment. Thereby must we differentiate between an active (congestive) and a passive (engorgement) hyperæmia. 1. The causes of the *active* form are usually those of acute nephritis, the first stage of which in very many cases represents congestion of the kidneys, from which it is not always easy to distinguish it. Thus it occurs, e.g., after the action of acrid food-stuffs upon the kidneys, among which must be mentioned the various kinds of anemone, and adonis, the asclepiades, also damp, mouldy oats, containing fungi, or hay in a similar condition. Active hyperæmia of the kidneys, with polyuria, its chief symptom, occurs sometimes from the same cause, and in spring especially is enzootic. Bad or dirty drinking water from puddles and marshes may also produce congestion of the kidneys. Indeed, any form of plethora or excess of blood in the abdomen brings more or less hyperæmia of the kidneys in its train, which under favouring circumstances may develop to haematuria. Finally, active hyperæmia of the kidneys is an accompaniment of many infectious diseases (influenza, murrain, rabies). 2. *Passive* hyperæmia of the kidneys follows on general engorgement of blood as a result of heart or lung affection (valvular defects, emphysema or thickening of the lungs, exudations of the pleura), as well as on engorgement of the posterior vena cava following on aneurisms, thromboses or tumours, or from abnormal accumulation of food or gas in the intestinal canal, or, finally, after thrombosis or compression of renal veins and renal arteries (continued thrombus from aneurism of the anterior mesenteric artery or artery of the kidneys).

Dissection shows that in both forms of hyperæmia the kidneys are enlarged, swollen, softened and highly reddened. In the active form it is rather the surface vessels which are filled, and in the passive the veins of the medullary substance. In active hyperæmia especially, the kidneys are saturated with moisture and very soft (parenchymatous oedema); the capsule is loose, and the upper surface of the organ sometimes spotted with blood in consequence of haemorrhage. In prolonged passive hyperæmia of the kidneys the organ takes on a dark, bluish red colour and becomes hardened by proliferation of the interstitial tissue (cyanotic induration). At the same time the epithelium of the tubuli uriniferi suffers disturbance of nutrition in the shape of fatty degeneration, to which haemorrhage may also be joined. Finally, passive hyperæmia may develop into nephritis.

**Symptoms.**—In active hyperæmia of the kidneys we find polyuria, with pale, thin urine of low specific gravity. The quantity may, in a horse, rise from about 4½ quarts a day to 5½ gallons and more, and the specific weight may sink from the normal 1040 to 1001-5 (a form of diabetes insipidus). The animals also hold their bodies in a slightly curved attitude and walk stiffly. There is no fever. In passive hyperæmia of the kidneys the quantity of urine is, on the contrary, diminished, and albuminuria sets in (even haematuria); hyaline casts also occur sometimes in the urine. There is no fever, but a slight weakness in the hind legs. The course is chronic. In active hyperæmia it is advisable to drain off to the intestinal canal; in the passive form adopt the treatment prescribed for the primary ailment (digitalis), active exercise, etc.

## 2.—CHRONIC INFLAMMATION OF THE KIDNEYS. NEPHRITIS CHRONICA.

(*Nephritis indurativa, fibrosa, interstitialis, sclerosis of the kidneys, atrophied kidney, cicatrized kidney, granulated kidney, granular atrophy of the kidneys.*)

**Etiology.**—Chronic inflammation of the kidneys either takes from the beginning a creeping, chronic course, or it develops from acute nephritis. Of the true causes of chronic inflammation very little is known. They must, however, be sought in the action of injurious irritants upon the tissues of the kidney. These irritants may be both parasitic and chemical in nature. The bacillus of glanders must rank first among the parasitical irritants. But not rarely is chronic nephritis set up by the intrusion into the blood of inflammatory bacteria and their elimination through the kidneys. Hereby both the bacteria and their products of decomposition may act as irritants upon the tissues of the kidney. This explains the frequent occurrence of this disease in cows, which, in consequence of the act of calving, are much exposed to the action of septic matter from the uterus. Other doors of entrance for the inflammatory agents are (in horses) ulcers in the bowels and larynx, as well as chronic bronchial catarrh with formation of bronchiectases and emphysema. Further, an embolic chronic nephritis sometimes occurs in horses in connection with an existing worm-aneurism of the anterior mesenteric artery and of the renal arteries with metastasis and formation of thrombi.

Senile vascular changes (atheromatous degeneration) may also be connected with chronic inflammatory processes in the kidneys in older animals, the same as in man. Finally, frequent chills may give rise to chronic nephritis.

The latter disease is very often complicated with chronic inflammation of the pelvis of the kidneys (pyelitis), especially in cattle. This chronic pyelo-nephritis of cattle, which is distinguished by a special irritant factor, will be treated in a later chapter.

**Pathological Anatomy.**—The various forms in which chronic nephritis manifests itself are, according to Kitt :

1. **Nephritis fibrosa multiplex**, also spoken of as *sclerosis maculata*, presents multiple deposits of connective tissue and contractions of the kidney, which are crater-shaped and occur mostly in cattle. On the upper cortical surface we see yellow, yellowish-white and greyish-white islands dotted about, which are slightly sunk or scabbed over in streaks, which on section contract into stripes. The kidney has a firm consistence and lumpy surface. Newly formed connective tissue, cicatricial tissue and cellular foci of infiltration are shown by the microscope. These all point to an inflammatory origin of the sclerosis (*sclerosis ex nephritide*), in contrast to the sclerosis embolica which arises after emboli (*nephritis fibrosa multiplex embolica*, scabbed kidney, *ren cicatricosus*).

2. **Nephritis fibrosa diffusa**, called also *sclerosis totalis* or *diffusa* in contrast to the former form, consists in a general and abundant interstitial proliferation of the connective tissue, which hardens the entire kidney, especially of cattle. The kidneys are abnormally large, extremely hard, white, yellowish-white or whitish-grey in colour and resemble a large lump of connective tissue without any lobular separation. It is difficult to cut, creaks under the knife and is formed of fibrous tissue alternated with chalky layers. Microscopically examined, the kidney consists of cicatricial tissue, very fibrous and made up of spindle-shaped and round cells, instead of the parenchyma of the kidney, of which latter nothing is left but canal-like spaces and empty clefts or stratified chalky substance. Between these are crater-shaped cellular infiltrations.

3. **Nephritis granulosa**, which must be regarded as a

chronic fibrous inflammation of the kidneys, with tendency to shrivelling (shrivelled kidney). The upper surface in this form is uneven, wrinkled, lumpy, rough, granular (nephritis granulosa, or granular atrophy of the kidneys), the consistence is firm, the colour, white or whitish grey and the capsule hard to cut. The shrivelled kidney of pigs is the product of countless, closely-ranked inflammatory foci.

4. **Nephritis fibro-vesiculosæ** is a complication of fibrous inflammation of the kidneys with cystic degeneration of the tubuli uriniferi, with formation of numerous small vesicles.

**Nephritis fibro-plastica.**—Kitt thus names a nephritis (also as nephritis maculosa alba, or white-spotted kidney of calves) frequently found in slaughtered calves and distinguished by whitish grey, spotted, somewhat prominent multiple foci, which are round and semi-spherical, varying from a millet-seed to a pea in size, and are wedge-shaped on section. The microscope shows in the white foci a plentiful accumulation of colourless cells, fibroblasts and connective-tissue cells.

**Glomerulo Nephritis.**—This is an accompaniment of various nephrites, both the parenchymatous and the different chronic fibrous forms, and is characterised by a preponderating inflammation of the Malpighian corpuscles.

**Nephritis mixta.**—A combination of interstitial inflammation and degenerative change of the epithelium, with slow course.

**Nephritis simplex.**—Simple cellular infiltration in the interstitia connective tissue, in the form of opaque blotches and streaky discolouration of the cortex.

**Nephritis urica.**—The renal gout of fowls consists in a uric-acid incrustation. The upper surface of the kidney is sprinkled with white prominent dots, the kidneys are much enlarged, hard and their section-surface marbled in yellowish-brown and red-brown.

**Symptoms.**—Chronic inflammation of the kidneys usually develops quite gradually without any definite symptoms. The animals are therefore far advanced in disease before submitted for treatment. The first striking signs are also of very general nature, such as loss of appetite and weariness, for which a superficial examination can find no organic cause. A characteristic indication of the parenchymatous stage is first obtained by the appearance of dropsical indications, viz.: swellings in the lower parts of the legs, of the breast and of the belly. These signs suggest examination of the urine, which alone can afford

a sure index to the nature of the disease. We find, then, the amount of the urine diminished, its specific gravity increased, the urine very rich in albumen and containing casts, epithelium, fatty granular cells, and sometimes also red blood-corpuscles. Further examination reveals a very hard and tense pulse, a strong heart-beat, which can often be felt on both sides, very loud cardiac sounds, as well as an occasionally marked cardiac dulness, which are all symptoms of hypertrophy of the heart. The temperature in the rectum is only slightly or, at most, moderately raised. Great thirst is generally manifest. If at this stage the intensity of the disease rapidly increase, the symptoms already described of uræmia show themselves, accompanied by extreme wasting and weariness, of which the animals quickly perish.

If from this parenchymatous stage chronic indurative (hardening) nephritis develop, the phenomena of the disease change entirely. The transition from the first form to the second is at first marked by a fall in the specific gravity of the urine, from 1025 to 1015 (in horses). In pronounced cases of atrophied kidneys the specific gravity is only 1001 to 1010; the urine is very watery and plentiful, contains but little albumen and few casts or epithelium. At the same time the œdema disappears. This is explained by increased hypertrophy of the heart, which, by causing greater blood-pressure, promotes absorption of the collected transudation caused by stoppage, thereby raising the quantity of urine, while lowering its specific gravity and proportion of albumen. Thirst is very intense.

If compensation on the part of the heart be sufficient, this condition may last for years. Such compensatory hypertrophy of the heart leads, however, sooner or later to gradual and increasing incapacity of that organ. The pulse grows feebler and irregular; and to complicate the growing vascular congestion in the body, bronchial catarrh sets in, with chronic catarrh of stomach and intestines, diffuse œdema, shortness of breath, palpitation, dizziness and even pneumonia, pleuritis, pericarditis, along with general haemorrhage of the organs. The urine again diminishes, while its specific gravity and albuminous contents increase, and death finally ensues under the aspect of an acute or chronic uræmia.

**Therapeutics.**—The treatment of chronic nephritis is, in the main, the same as that of the acute form, but the prospects of cure are slighter. Besides this, it is chiefly a question of

improving the general condition of nourishment and removing the anaemia; also, in cases of heart failure, of increasing the blood-pressure. Administer therefore, first of all, cardiac tonics and diuretics, such as digitalis, diuretin and caffein, tincture of strophanthus and also calomel. Pilocarpine and arecolin are also to be used here if dropsy be present. As a resorbent for the hyperplastic tissue in the kidneys, iodide of potassium may be recommended.

**Relation of Chronic Nephritis to Hypertrophy of the Heart.**—There are various opinions on this subject.

1. According to Traube, the blood-pressure is raised by the accumulation of water in the blood and the disturbance of circulation consequent on changes in the liver, the result of which is a compensatory hypertrophy of the heart. 2. In opposition to this mechanical theory is Senator's chemical theory, according to which the increased blood-pressure is caused by retention of the urinary constituents in the blood, and particularly of urea. At present it is impossible to decide between these two theories.

**III.—PURULENT INFLAMMATION OF THE KIDNEYS. NEPHRITIS PURULENTA.**

(*Abscess of the kidneys, Embolic purulent Nephritis pyonephrose.*)

**Etiology.**—The pathogenic agents of purulent nephritis reach the renal tissues in two different ways, from the blood (haemogenous) or from the bladder by the ureters (urogenous). Purulent, ichorous, and, indeed, any infective emboli produce local inflammatory foci, as in septicæmia, endocarditis, gangrene of the lungs, pneumonia, pharyngitis, etc. Emboli of non-infective nature, such as those arising from worm-aneurism, produce no purulent nephritis, but an ordinary chronic one, with congestion and formation of new growth. Purulent nephritis, in the form of abscesses in the kidney, is most common in cattle. Here certainly one has to reckon with septic infection caused by hard labour-pains, or retention of the after-birth, with deposit of bacteria in the excretory organ of the kidneys. *From the bladder*, inflammation may spread by way of the ureter, first to the pelvis of the kidneys, and thence to the kidneys themselves (pyelo-nephritis). Finally, suppuration in the pelvis of the kidney may be caused by traumatic influences, for instance, by renal calculi.

**Pathological Anatomy.**—According to Kitt, purulent nephritis occurs in domestic animals, chiefly in two forms.

1. *Diffuse Purulent Nephritis* arises when the pyogenic matter is distributed by the blood over the entire kidneys (*nephritis purulenta punctata, diffusa, mixta*). The whole capsule is permeated by numerous small abscesses in the shape of pus-coloured, whitish-yellow dots and streaks, which are surrounded by a bright red area and dark red haemorrhages. The sectional surface of the kidney displays a bright appearance, sprinkled with white, grey, yellow, brown and red. These small abscesses in the cortex have their points towards the boundary and medullary layer; they contain numerous bacteria, especially clumps of micrococci. This disseminated form of purulent nephritis occurs chiefly among pigs and calves.

2. *Nephritis apostematosa, or abscess of the kidney*, occurs either embolically, as in horses during the progress of strangles, or urogenously, as in cattle during purulent pyelo-nephritis. We find in the kidney purulent cavities, either singly or in numbers, from the size of a hen's egg to that of a man's head, with creamy pus and speckled-white borders, in which (in strangles) the presence of the streptococcus of strangles can be demonstrated. In other cases the whole kidney is changed, after destruction of the parenchyma, into a fluctuating abscess, the walls of which consist of the capsule of the kidney and the indurated cortical substance, whereby the extended pelvis of the kidney is drawn into the abscess-cavity (*pyo-nephrosis*).

**Symptoms.**—These are not very characteristic, apart from the fact that they are masked by the symptoms of the primary disease (*pyæmia, pyelitis, etc.*). Often only great emaciation, disturbed appetite, increase of thirst, weariness, roughness of coat, etc., can be noticed. In other cases the urine offers some points for guidance. Thus it sometimes smells badly, is of thick, slimy consistence, mixed with pus and worm-shaped coagulae; also blood and dead kidney-tissue may be found in it. In cows the tail is found smeared with pus and mucus. In later stages embolic pneumonia, *pyæmia*, haemorrhagic diathesis, etc., may occur. But the course is generally slow and chronic, so that the changes in the kidney are not known until after slaughter. Occasionally the presence of soft, fluctuating abscesses can be recognised in cattle by exploration of the abdominal cavity through the rectum.

**Therapeutics.**—Treatment is purely surgical, and may consist either in puncture of the pyogenic membrane or abscess from the lumbar region, or in extirpation by laparotomy. The latter operation was tried on a cow by Münich. Though in this case the result was not very satisfactory, yet this operation may nevertheless be recommended, seeing that cows are not very sensitive to such operations and, if unsuccessful, may still be killed without much pecuniary loss. Maksutow claims to have removed the kidney from a horse with success.

#### INFLAMMATION OF THE PELVIS OF THE KIDNEY. PYELITIS.

**General Notes.**—This complaint offers more of interest to the pathological anatomist, than to the clinicist. For one thing, it always occurs as a secondary condition, consequent on another primary disease (nephritis, cystitis), and then it can only rarely be with certainty diagnosed during life. A detailed discussion of pyelitis would therefore scarcely be fitting here.

**Etiology.**—Inflammation of the pelvis of the kidneys usually results from the spread of inflammation from the bladder or kidney to the mucous membrane of the pelvis (pyelo-cystitis and pyelic nephritis). It also often develops in the course of infectious diseases and of poisoning cases, when the infectious or poisonous matter comes in contact with the mucous membrane of the pelvis of the kidney on being excreted from the body. We may here remind our readers of glanders and tuberculosis, as well as of the action of acrid vegetable poisons. Moreover, foreign bodies, especially renal calculi, cause inflammation—at times slight, at times very severe—in the mucous membranes just named (pyelitis calculosa). The lodgement of gravel or sand in the pelvis of the kidneys may also produce pyelitis. We must notice, too, the presence of the giant palisade worm (*eustrongylus gigas*) in dogs, horses and cattle. Accumulation of urine in the pelvis of the kidney, after mechanical or dynamic retention, likewise produces pyelitis as a result of the decomposition of the urine. If such retention last long, atrophy of the kidney may develop, with cystic breaking down of the renal substance (hydro-nephrosis).

**Pathological Anatomy.**—According to the degree, cause and duration of the inflammation, we may distinguish between simple, purulent, croupy, diphtheritic, calculous and also acute

and chronic pyelitis. In the simple form the mucous membrane is swollen, reddened, sometimes permeated by haemorrhage, as well as covered by a coating of mucus, rich in desquamated epithelial cells, which, in purulent pyelitis, consists almost entirely of white blood-corpuscles. The effect of renal calculi upon the mucous membrane manifests itself either by the occurrence of circumscribed, inflammatory foci, progressing sometimes to necrosis and formation of ulcers, and of croupous-diphtheritic deposits; or else it is shown in the form of an incrustation, with inflammation of the mucous membrane, in which the excreted sediment combines with the mucous membrane to form a rigid coating. Chronic catarrh of the pelvis, so common in horses, is revealed in a dirty brownish discolouration of the kidney, with pigmentation, thickening, submucous infiltration and ulceration of the mucous membrane, accompanied by enlargement of the pelvis. Pyelitis soon becomes complicated with nephritis or cystitis, and the purulent form leads especially to purulent nephritis and pyo-nephrosis. *Eustrongylus gigas* is, in isolated cases, found to have wandered from the pelvis of the kidney into the abdominal cavity near the kidneys.

**Symptoms.**—The symptoms of this form of inflammation are very indefinite, owing to frequent complication with nephritis and cystitis. Pyelitis can only then be with certainty diagnosed when, on microscopic examination of the urine, the characteristic epithelial cylinders fitted with processes (shaped like molar teeth) are found, such as are peculiar to the lower layers of the mucous membrane of the pelvis. Sometimes in horses an enlargement of the pelvis, with thickening, i.e., widening of the urinary channel may be demonstrated by palpation of the kidneys from the rectum. The same, too, in dogs by palpation of the abdomen. All other symptoms agree with those of the primary disorder. Thus the presence of renal calculi produces colic and difficulty of urination (so-called kidney-colic), and in long retention of urine may lead to uremia. *Eustrongylus gigas* caused symptoms in two dogs which led to suspicion of rabies. In the urine one finds, according to the type of pyelitis, pus corpuscles, coagulae of blood and fibrin, necrotic mucous tissue, sand, and gravelly lumps and numerous strongylus ova, if *eustrongylus gigas* be present in the pelvis.

**Therapeutics.**—The treatment of pyelitis corresponds

with that of the primary disease. It consists chiefly in administration of anti-catarrhal, antiseptic and astringent remedies. To these belong the alkalis, boracic acid, salicylic acid, creolin and all those remedies which contain tannic acid.

## PYELO-NEPHRITIS IN CATTLE.

**Etiology and Pathogenesis.**—Among inflammatory disorders of the kidneys pyelo-nephritis occupies an independent position in so far as it is caused by a special infectious agent, the bacillus renalis bovis (*bacillus pyelo-nephritidis bovis*). This bacillus, according to Enderlen and Höflich, who simultaneously discovered it, forms immovable, slender or slightly curved rods, with rounded angles, which lie in heaps together and can easily be stained by Gram's method. The bacillus, which flourishes best at a temperature of 98.6° Fahr., forms on a firm nutrient ground, colonies which are grey, dotted, protuberant and very lustrous, and in bouillon a fine granular sediment.

Bacillary pyelo-nephritis does not seem to be a rare form of disease among horses, as we know from observations made in the slaughter-houses. The fact that it occurs mostly in mares and appears oftenest not long after parturition, points to some connection with septic processes in the uterus, and especially to some retention of putrid after-birth. The intrusion of the bacillus from the womb to the kidneys probably happens often by urogenous means, i.e., through the vagina, urethra, bladder and ureter. The probability of such an ascensive inflammation is confirmed by the fact, that vaginitis, urethritis and cystitis are actually often present along with pyelo-nephritis. Moreover, the anatomical position of the orifice of the urethra on the under side of the vagina favours the access of micro-organisms to the bladder. On the other hand, bacillary pyelo-nephritis may be caused by haematogenous means, as experiments in intravenous transmission have shown that blood can convey the disease.

**Pathological Anatomy.**—The bacillus of pyelo-nephritis causes pus formation and necrosis in the kidney. Around the inflammatory foci interstitial proliferation of connective tissue is produced in the later stages, along with sclerosis. We find sometimes one and sometimes both kidneys affected, and usually enlarged to double the normal size. The

capsule of the kidney is often opaque and in places adherent. The upper surface of the kidney has a spotted appearance, in which clay-coloured or greyish-yellow circumscribed foci, from a pin-head to a pea in size, or even larger, alternate with dark red patches. Occasionally, too, this surface is lumpy and covered with numerous small abscesses. The substance of the kidney is harder than usual. On section we find yellowish grey, yellowish white or white streaks in the cortical substance, and at times also minute abscesses arranged in rows; similar light grey stripes are also found in the medullary substance. The renal papillæ are often covered with a dirty yellow purulent substance, or else are ulcerated and necrotic (necrotic papillary nephritis). The pelvis of the kidney is enlarged and contains dirty grey or yellowish brown matter, which is purulent, mucous, flaky, crumbly or thinly fluid. Its mucous membrane is thickened, spotted with red, rough and occasionally covered with ulcers or warty proliferations. The urethra often shows similar changes and is usually three to five times its normal thickness. Microscopical examination of the tissue of the kidney reveals minute cellular infiltration in the cortical substance, increase of interstitial tissue in cortex and medulla, atrophy of the urinary tubules, obliteration of the glomeruli, as well as swelling and destruction of the epithelium of the tubuli uriniferi. In the pelvis of the kidney are numerous triple-phosphate (coffin lid) crystals, pus corpuscles, rod-shaped and flat epithelial cells and detritus, as well as yellowish-brown and greyish-brown collections of bacteria. At the lower end of the urinary tubules many bacteria may also be discovered by staining.

**Symptoms.**—The course of pyelo-nephritis being chronic, its symptoms are generally rather uncertain. Animals manifest a gradually increased disturbance of general health, beginning with lessened appetite and rumination, and leading on finally to great emaciation. This process is interrupted at times by intermittent colic and intermittent fever. Many cows stand with back arched and sensitive to pressure. Uræmic appearances, such as retention of urine, vomiting or colic, are rarer. The most important changes are to be found in the urine. In advanced stages of the disease this is mucous, purulent and even bloody. In consequence of this the surroundings of the pudenda, especially the tail, are often smeared with putrid and bloody matter. Under the microscope we detect

in the urine the presence of white and red blood-corpuscles, rod-shaped and flat epithelial cells, triple-phosphate crystals, numerous bacilli and albumen. Examination of the kidneys through the rectum generally yields a negative result. Not seldom is the aspect of the disease modified by complication with cystitis, metritis and vaginitis. As an indication of catarrh of the bladder we must notice: frequent urination, sometimes even strangury. A simultaneous affection of the uterus and vagina reveals itself by a purulent, mucous or bloody discharge quite independent of urination.

**Differential Diagnosis.**—This chronic pyelo-nephritis can only with certainty be clinically distinguished from the chronic nephritis (atrophied kidney) already described when the presence of bacilli can be proved in the urine. This applies also to simple pyelitis, especially its calculous form. Without such proof pyelo-nephritis is difficult to distinguish from simple nephritis. If, however, an attempt at cure must be made, give internal antiseptics, in order to disinfect the diseased parenchyma of the kidney. Of such we would name: camphor, oil of turpentine, salicylic acid, chlorate of potash and folia uvæ ursi. Camphor is usually given as an emulsion with linseed decoction; folia uvæ ursi as a decoction. In addition to these the remedies prescribed for nephritis may also be used.

**Amyloid Kidney.**—Amyloid degeneration of the kidneys has hitherto been only rarely met with among our domestic animals as an independent disease. It occurs more frequently along with amyloid degeneration of other organs, and is more or less subordinate. Rabe noted it as an apparently independent ailment in three dogs, and Gerlach in an ox. The *anatomical changes* consist in a wax-like hardness of the cortical parenchyma, which is pale, dry and poor in blood, enlargement and glassy swelling of the glomeruli, together with the presence in the tortuous tubuli uriniferi of homogeneous, dullly lustrous urinary casts, which slightly refract the light. Treated with methyl-violet, these parts take on an intense purple red, and with a watery solution of iodine in iodide of potassium a mahogany red, which last changes to steel-blue on addition of sulphuric acid. According to Rabe's observations, the phenomena consist in dropsical swelling of the subcutis, especially of the limbs, in ascites, albuminuria and in uræmic symptoms, viz., vomiting, weakness like that of paralysis, coma and fall of bodily temperature. No hypertrophy of the heart was in any case noticed. The *treatment* of amyloid kidney is the same as that of chronic nephritis, with which, during life, it is often confounded.

**Tumours of the Kidney.**—These are but rarely subjects of clinical

investigation and treatment. In some cases, with small domestic animals, especially dogs and cats, these tumours (carcinomata, sarcomata, melanomata, adenomyomata, sarcomyomata and rhabdomyomata) may be clinically demonstrated by palpation of the abdomen. Generally, however, they are only revealed by dissection, when, during life, haematuria, sensitiveness in the renal region and disturbed urinary secretion have been observed. Treatment would merely consist in extirpation of the diseased kidney.

**Coccidiosis of the Kidneys.**—Railliet and Lucet noted a case of liver complaint produced by coccidia in the kidneys (compare p. 261).

#### RETENTION OF URINE—RETENTIS URINAE.

**General Remarks.**—This consists in an abnormal accumulation of urine in the bladder, with consequent distension and inflammation of the same, as well as of the ureters and pelvis of the kidneys, which leads eventually either to rupture of the bladder and peritonitis, or to atrophy of the kidney (due to pressure), to hydro-nephrosis and uræmia. Nevertheless, it is not an independent disease, but merely a symptom of various diseases. It occurs most frequently in cattle, dogs, horses and sheep, and chiefly in the male (*i.e.*, the castrated) animals, only rarely among females.

**Etiology.**—The causes of this retention are as follows:—

1. *Foreign bodies in the bladder and urethra*, which hinder the flow of urine. These include cystic and urethral calculi, urinary sediment in the S-shaped bend of the urethra in oxen and wethers, broken pieces of catheter, or foreign substances purposely introduced into the urethra (straws, bits of broom), blood-coagulae or particles of fibrin, portions of croupy and diphtheritic membrane in the bladder and polypoid tumours which cover up the orifice of the bladder.

2. *Compression with obstruction of the urethra and orifice of the bladder by adjacent new growths*, especially of the prostate. Then compression of the uterus, ovaries or of the bladder itself by accumulation of dung in the rectum, displacement of the uterus, pregnancy; narrowing of the lumen of the urethra by stricture, inflammation or swelling, kinking of the penis, fracture of the os penis, paraphimosis (strangulation of the glans penis by swollen foreskin), phimosis (abdominal narrowing or obstruction of the orifice of the prepuce).

3. *Paralysis of the Urinary Bladder* (detrusor urinæ), in the form of a paresis or paralysis following upon severe

croupous, diphtheritic, phlegmonous cystitis, pericystitis, affection of the spinal medulla (traumata, meningitis spinalis, apoplexy) or general weakness, after too prolonged a suppression of urination in horses suffering from brain affection, or which have been too long in movement, and lastly after reflex paresis of the muscular apparatus of the bladder during colic in the course of rheumatic haemoglobinæmia of horses and in severe general disorder.

4. *Spasm of the Sphincter* (*sphincter vesicæ*) in chills, cystitis or when foreign bodies lodge in the bladder; also, in reflex manner, in colic and tetanus.

**Urinary Calculi.**—With regard to the development of these, which are of great surgical importance, we may here observe that they can have a three-fold origin. They are either formed by the salts contained in the urine crystallizing around some foreign body in the bladder (coagula, sperma), or they are due to an excess of salt in the urine caused by drinking water containing too much lime, or eating food too rich in lime and magnesia (e.g., bran), or excessively nitrogenous fodder (producing uric acid); or finally, to formation of insoluble phosphates of ammoniacal magnesia (triple-phosphate) in cases of retention and decomposition of urine in the bladder (cystitis). According to Ebstein's experiments, inflammatory processes in the urinary passages pre-dispose to the formation of calculi by supplying the organic foundation for them. In their chemical composition the calculi of herbivorous animals are mainly carbonates (also oxalate and silicate calculi); those of the carnivora contain chiefly salts of uric acid (along with oxalate and cystine calculi); and those of the omnivora oxalates and carbonates. In all three kinds of animal, phosphatic calculi (phosphate of ammoniacal magnesia) are not rare, occurring after retention and decomposition of urine in the bladder. By giving a dog four to six grains of oxamide daily, Ebstein and Nicolaier succeeded in producing artificial calculi in a few weeks.

**Symptoms.**—These show themselves either in an entire suppression of urination (ischuria) or an imperfect and difficult passage of water accompanied by pain (dysuria, strangury). An accumulation of urine in the bladder produces in the more sensitive animals, such as horses and dogs, very early signs of discomfort and pain, whereas, in the indolent ruminants, especially oxen, several days may elapse before any characteristic symptoms are noticed. Horses and dogs show at first the symptoms of colic (so-called urinary colic), with frequent attempts to micturate. In cattle and sheep such signs of colic are often entirely absent, though perhaps not rarely overlooked, or we observe only a peculiar peristaltic movement and twitching on the peritoneum along the course of the urethra,

and also the unusual *ness* of the orifice and its terminal tuft of hair. Palpation of the bladder from the abdominal walls or rectum shows that it is very full—unless rupture has already occurred—and the animal evinces pain on its being pressed. The presence also of some obstruction may, at the same time, be demonstrated, such as a calculus, new growth, etc. This may be further confirmed by careful examination of the orifice and course of the urethra, or by introduction of a catheter or sound. In addition to the above symptoms, the animals seem spiritless, cease to eat or drink, their pulse is quickened, and sometimes they break into profuse sweat.

If at this stage no relief be procured, the symptoms increase and result either in rupture of the bladder with rush of urine into the abdominal cavity, followed by peritonitis; or in diphtheritic inflammation of the bladder, which develops into pyelitis, nephritis or hydro-nephrosis and uræmia. In the former case the signs of pain and disquiet suddenly cease. On exploration the bladder is found quite empty, while the presence in the abdominal cavity of much liquid can later be detected. Along with great dulness of the sensorium, the pulse and temperature both rise; shiverings and general muscular tremblings appear; both breath and sweat acquire a distinct urinary smell; the abdomen grows ever fuller; urination ceases entirely, so that at last the animals perish from diffuse peritonitis combined with uræmia. Horses and dogs die very quickly, in a few hours, after previous ischuria, lasting one or two, or, at most, three days. Cattle, on the contrary, may lie for eight to fourteen days after the bladder has burst, and have even been known to live four or five weeks. In the latter case the sickness ends with the phenomena of intense gangrenous cystitis, complicated with acute nephritis and uræmia. This termination is, however, less frequent.

**Differential Diagnosis.**—Retention of urine is as often mistaken for true colic as this latter for the former, especially by the non-professional. This is not surprising, seeing that true colic is frequently accompanied by a kind of urinary retention and that the bladder, on examination, is generally found more or less full. Yet a differentiation is, with a little care, not difficult. In true colic the symptoms of a bowel-complaint gradually predominate, while in retention of urine those of an affection of the bladder become prominent in the form of strangury, frequent adoption of the attitude

of urination, spreading the legs, active wagging and lashing of the tail, persistent ischuria or strangury and sensitiveness of the bladder to pressure. There are also other diseases which may give rise to error, e.g., rheumatic haemoglobinæmia of horses, simple cystitis, nephritis, peritonitis and finally labour-pangs. But in none of these is the discharge of urine permanently disturbed, nor the bladder for long abnormally full.

**Therapeutics.**—In the mechanical form of urinary retention, the treatment is entirely surgical (urethrotomy, cystotomy, puncture, circumcision). In the so-called dynamic form, caused by paralysis or cramp of the bladder—more frequently by the former—one must attempt first of all to empty the bladder by pressure from the abdominal wall or from the rectum, as well as by introduction of a catheter. Reflexly one may also secure this result by irritating the orifice of the urethra, or by gently stroking the urethra along the perineum. If there be spasm of the sphincter, we recommend plentiful rubbing of turpentine, or the oils of aniseed, caraway, fennel and camomile upon the abdomen and perineum, as well as internal administration of these oils, together with morphia, belladonna and chloral hydrate. Strychnine, physostigmine and electricity may also be tried in severe paralysis of the bladder.

**Incontinence of Urine.**—This is a symptom of several complaints, especially of affections of the spinal medulla, and springs from paralysis of the sphincter vesicæ, only rarely from cramp of the detrusor, e.g., in cystitis. The condition is also noticed after prolonged retention of urine; in weakness of the sphincter from old age and in cystitis; after cystotomy, when the muscle of the sphincter has been severed; when it has been destroyed by tumour; after destruction of the bladder by stones or of its orifice by polypi. The treatment must be regulated by the primary disease. In paralysis of the sphincter it consists in administration of strychnine, the application of electricity, cold rinsing of the bladder, and general strengthening of the constitution.

#### CATARRH OF THE BLADDER.—INFLAMMATION OF THE BLADDER. CYSTITIS.

**General Remarks.**—These two complaints, as indeed, bladder-affections in general, belong to the realm of surgery. If they are briefly cited here, it is because all such affections are closely connected with those of the kidneys, whose symptoms

they often much resemble, so that the treatment of bladder-disorders is partly internal. Moreover, the retention of urine just described is, when long continued, always complicated with cystitis.

**Etiology.**—The causes of cystitis are first irritation of the mucous membrane of the bladder by poisonous and infectious matter, which has been excreted from the blood, such as cantharides, turpentine or bacteria. Under this head comes also the inflammation of the bladder occurring in the course of severe infectious disease. Chill may also be a possible cause. Then, too, cystitis arises from complication with inflammation of an adjacent organ, thus from nephritis, pyelitis, urethritis, vaginitis, peritonitis or metritis. Traumatic influences, such as bruising or crushing the mucous membrane by calculi, by sounds, or by infection through an unclean catheter, may all produce cystitis. Finally, retention of urine in the bladder, as by a dog when kept too long in a room, may cause cystitis, at first catarrhal, but at last necrotic. Compression, stricture or stenosis of the urethra (paralysis of the detrusor), cramp of the sphincter consequent on rapid decomposition of the urine and the action of its septic matter upon the mucous membrane may all produce the same result. Among bacteria, the bacterium coli commune and also proteus vulgaris are especially connected with the etiology of cystitis.

**Post-mortem Lesions.**—These vary according to the degree of the condition. As with other mucous membranes, so here, too, an inflammation may be either catarrhal, ulcerous, croupous, diphtheritic, necrotic or haemorrhagic in nature; it may be chronic, hypertrophic, associated with varicose degeneration of the vessels; or phlegmonous, leading on to submucous and perivititic abscesses, even to perforation of the bladder; or, finally, it may become a so-called "incrusting" inflammation, that is to say, complicated with deposit of urinary salts in the mucous membrane of the bladder. In the simple catarrhal form we find the mucous membrane swollen, reddened and covered with a layer of mucus, cast-off epithelial cells, with occasional white blood-corpuscles, in the ulcerous form, this layer consists almost entirely of white blood-corpuscles. The chronic form is distinguished either by thickening, by warty proliferation, varicose enlargement of the vessels of the mucous membrane, or—as is frequent in mares and

cattle—by incrustation of the same with lime-salts. In the croupous and diphtheritic forms, membranous deposits and necrotic scurf overlay the mucous membrane, leaving ulcers behind them.

**Symptoms.**—In an acute cystitis the urine is passed frequently, in small quantities, and even in drops, causing more or less pain to the animal. Male animals straddle widely, erect the penis and draw up their testicles. Females press hard, open and shut the orifice and move the clitoris. Unrest and colic are also apparent. Palpation of the bladder from the outside (in small animals), or from the rectum is painful. According to the degree of inflammation there is more or less fever, consumption of food is interrupted or ceases entirely. But the greater changes are found in the urine. This contains products of the inflamed mucous membrane: desquamated epithelial scales, white blood-corpuscles, mucus, albumen and, where decomposition of urine has set in, crystals of ammonio phosphate of magnesia and bacteria. With the carnivora the acid reaction changes to a neutral or alkaline. Finally the urine shows—and most distinctly in purulent cystitis—a more or less thick, slimy or flaky sediment, which is at times entirely purulent. In the croupous diphtheritic form necrotic pieces of tissue are mixed with this; and in the haemorrhagic form red blood-corpuscles.—*Chronic cystitis* runs its course not unfrequently without any striking external signs, and is consequently often overlooked, especially in cattle. Examination of the urine is therefore important for diagnosis, both here and in all kidney diseases.

**Therapeutics.**—The internal treatment of cystitis consists in administration of boracic acid, salicylic acid, benzoic acid, chlorate of potash, tannin, naphthalin, resorcin and creolin. Of the last-named, give to a moderate-sized dog daily, three minim doses. In chronic cases, give internally resinous remedies, viz., turpentine, balsam of copaiba and balsam of Peru. Trasböt also recommends for catarrh of the bladder, bromide of potash and bromated camphor. The chief treatment of cystitis is, however, external, and consists in rinsing out the bladder with antiseptic fluids, e.g., with a  $\frac{1}{2}$  per cent. solution of creolin, or 1 to 3 per cent. of carbolic acid, boracic acid, chlorate of potash, as well as a 1 to 5 per thousand solution of

sublimate, or  $\frac{1}{2}$  to 2 per cent. of nitrate of silver. For all further instructions, see the text-books on surgery.

**Hæmorrhage of the Bladder.**—The causes of this are either injuries by calculi, contusion by a fall, pressure consequent on fracture of the pelvis, or some inflammation of the mucous membrane of an ulcerous or hæmorrhagic nature (*cystitis hæmorrhagica*). Acute inflammation caused by chemical irritants, and chronic inflammation concurrent with hæmorrhoidal enlargement of the veins are very common causes. Finally, new vascular growths, in process of suppuration or ulceration, especially papillomata, sarcomata and carcinomata. The *symptoms* consist in admixture of blood in the urine (haematuria), which stains it and forms therein a red sediment composed of blood-corpuscles and fibrinous coagula. Sometimes more blood is noticed after previous severe exertion, when, occasionally, almost pure blood may be passed. In consequence of the decomposition of this blood, catarrhal inflammation of the kidneys usually supervenes if the hæmorrhage be long continued. General health is also much impaired, being marked by growing anaemia and weakness. In fact the patient not seldom bleeds to death. The treatment of hæmorrhage of the bladder is mainly external and surgical. It consists in washing the bladder out with iced water, as well as with solutions of liquor ferri, creolin, alum and tannin. Along with these we may administer internally ergot, fluid extract of hydrastis, tannin, sugar of lead, etc. It is also advisable to guard against decomposition of the blood in the bladder by internal use of boracic acid, salicylic acid, creolin, chlorate of potash, etc., or by rinsing with disinfectants.

**Hæmaturia**, or bloody urine, which is the passing of red blood-corpuscles with the urine, must be distinguished from hæmoglobinuria, which is an admixture of the red colouring matter alone (hæmoglobin). The fact that of late years they have been often classed together has made the differentiation and identification of certain diseases of horses and cattle more difficult. Hæmaturia is certainly no uniform or independent disease, but merely a symptom of the most varied disorders of certain organs. As its causes we would indicate : 1. *Injuries to the kidneys* from a blow, heavy fall or contusion, over-exertion, or the intrusion of some foreign body. 2. *Inflammation of the kidneys* (hæmorrhagic nephritis); acute inflammations especially, often exhibit slight hæmaturia in their initial stage. Here, also, we may include those nephrites which are excited by animal or vegetable irritants (fresh fir-tree sprouts, cantharides, mercury, bryony, or sharp diuretics), by certain fungi (those of mildew, ergot or mould), as well as by some mineral poisons (mercury, phosphorus). First of all in practical importance is that form of hæmaturia caused by acid plant-stuff, a disease in cattle known generally as

"bloody urine" (see enzoötic gastro-enteritis). 3. *Pyelitis*, especially the purulent form; also the presence of *eustrongylus gigas* may produce haemorrhage. 4. *Cystitis*, both haemorrhagic and chronic, or the kind which causes enlargement of the vessels. Haemorrhage of the bladder occurs very commonly among cattle. 5. *Cystic calculi*, injury of the bladder, cystic-tumours (cancer, polypi, tuberculous and mucous infiltrations), ulcers in the bladder. 6. *Acute infectious diseases*, in the course of which so-called haemorrhagic diathesis—lops, especially anthrax, pyæmia and septicæmia, murrain, pox, distemper of dogs and petechial fever. 7. *Inflammation of the urethra*, urethral calculi. 8. *Prostatitis*, concretions of the prostate. 9. *Constitutional ailments*, in the first place haemophilia, also leucaemia, pernicious anaemia, etc. 10. *Filariae in the blood*, producing lesions of the renal vessels, with consequent haematuria.—The chief symptoms of haematuria are the red or reddish-brown colour of the urine, or red sediment in the same, and microscopical proof that red blood-corpuscles are present. In the matter of *differential diagnosis*, haematuria must not be confounded with flow of blood from the vagina or uterus (menstruation, metrorrhagia), nor with abnormal discolouration of the urine by medicinal pigments (rhubarb, senna, tar, biliary pigments, santonin, anti-febrin).

#### APPENDIX TO DISEASES OF THE KIDNEYS.

##### HÆMOGLOBINÆMIA OF HORSES.

*Black Disury, Azoturia, Lumbago, Hæmoglobinuria.*

**General Remarks.**—Hæmoglobinæmia of horses, as its name implies, has nothing to do with kidney diseases; for the mere fact that, during its course, a secondary nephritis is sometimes developed, can be no justification for so classifying it, as some have erroneously done. It ought really to be discussed with the muscular diseases. If, therefore, we speak of it here, this is done because the practitioner is accustomed to refer for so-called black disury among the kidney diseases, and also because a very important symptom (hæmoglobinuria) can be best treated in this group; and for the further reason, that we do not wish to separate black disury (or ischury, as it is also called) of horses from the so-called "bloody urine" of cattle, which may be in its nature, either a hæmoglobinæmia

or a nephritis, and therefore more nearly allied to the kidney diseases.

**Historical Notes.**—Hæmoglobinaæmia of horses has long been known under a multitude of names, such as black disury or ischury, lion-cramp, azoturia and many others merely of local application. It is certainly one of the oldest of all known diseases of the horse. Yet even to-day, the nature, and especially the etiology, of the complaint is not fully explained.

**Etiology and Pathogenesis.**—We first notice an inflammation of the muscles of the hind limbs, produced by cold, whereby, amongst other things, a dissolution of the muscular colouring matter (identical with hæmoglobin), and its passage into the blood, take place. We therefore consider Fröhner's name of "rheumatic hæmoglobinaæmia," as by far the most suitable for most cases of the disease. We would not, however, deny the possibility of an "infectious," or "toxæmic" form in horses. These latter are nevertheless rarer.

The commonest cause of hæmoglobinaæmia in horses is probably a preceding chill. Horses acquire a pre-disposition to chill, by standing some days in warm, steamy and ill-ventilated stables, being well fed the while, for instance, during holidays, or when lame. Heavy, hard-working horses are most affected. According to Goring, the complaint may be experimentally produced by chill. The warm and well-protected stables of richer owners are proportionately more troubled by this disease than the badly appointed ones of the poorer classes, and its appearance in a district can generally be easily traced to the varied stable arrangements. When animals that are richly fed and have been long kept in heated stables are brought out into the cold air, they are much more susceptible to chill than others living amid hardier surroundings.

The manner in which the cold acts upon the organism and sets free the hæmoglobin in the body, may be thus explained. As is shown by physiology, metabolism in the muscles increases on any irritation of the sensitive, temperature-nerves of the skin. If then intense irritants be set to act upon the skin, this change of substance in the muscular system becomes greater and may grow to disintegration of the organic albumen, that is, of the muscular substance itself. In consequence of this degenerative muscular change, the products of such metabolism

pass into the blood and, with them, also the colouring matter of the muscles. This colouring matter is identical with haemoglobin. Hæmoglobinæmia is therefore of myogenous, and not of hæmotogenous, origin. The startling effects of such a chill are explained by the fact of the animals having been rendered unfit to encounter it by days spent in a warm atmosphere. The reason why the hind-legs are chiefly affected is that they, with the croup and loin muscles, have most to do with a horse's movements, and are the parts least covered up to protect them from cold (Fröhner).

In a few cases the agency of chill seems to be absent, although, as is known, horses may take cold when standing in their stalls. For lack of a more definitely-known cause, these may be termed "infectious," that is to say, "toxæmic," hæmoglobinæmia.

In support of the above theory the following may be cited:—Chill is acknowledged by the great majority of observers to be the chief cause of the disorder. But it cannot be supposed to be by direct separation of the red blood-corpuscles that it acts, seeing that the degree of cold experienced is far less than that required for the disintegration of blood (freezing). From Lassa and Nassaroff's experiments it has been shown that parenchymatous muscular degeneration, with reduction of colour in the muscles of the skeleton, may be effected by sudden chill. It is known that in horses intense myositis occurs after chill, or during severe winter cold. Moreover, a decided "cold-disease," the so-called "paroxysmal or winter hæmoglobinuria," occurs in man, which is caused entirely by cold and closely resembles black dysury. According to Rosenbach, it may be experimentally produced by cold, e.g., by a cold foot-bath, and its symptoms coincide remarkably with those of the above-named ailment. Besides all this, Schindelka has proved by haemometrical methods that in rheumatic hæmoglobinæmia the blood contains much more haemoglobin than is the case with sound horses. This can only be explained by the fact that it has absorbed a quantity of other similar colouring matter, which can be none other than that of the muscles, which is identical with haemoglobin.

To all the other explanatory theories which have been advanced very serious objections may be raised, with which we will not here trouble our readers.

**Post-mortem Lesions.**—The chief changes found in horses which have succumbed to hæmoglobinæmia are in the muscles and blood.

1. *The muscular changes*, which are found chiefly in the muscles of the croup and thigh, consist, when viewed by the naked eye, in oedematous swelling and a paleness of colour. Microscopically viewed, they show a granular opaqueness, a

splitting up into layers, hyaline degeneration and loss of transverse striation. All muscles are not equally affected, and not always those of the psoas and croup alone. Thus, Siedamgrotzky found the bodily muscles in one case decolourised and degenerated, as follows :—First of all the loin or psoas muscles, the thin adductor, the posterior part of the other adductors of the thigh (fish-bone muscle), the large and broad thoracic muscles ; in the second place, the four-headed extensor of the lower leg, and in the third place, the anterior portion of the adductor of the hind thigh, its abductor, the gluteus muscle and the longissimus dorsi. As to their nature, these muscular changes must be regarded as an inflammatory degeneration of the fibres. Similar changes are sometimes perceived in the muscles of the heart. Occasionally small haemorrhagic foci are found between degenerated muscular fibres.

2. The most important changes in the *blood*, are that it has lost its normal colour and acquired an appearance like that of varnish or tar, and refuses to coagulate, or does so badly. Blood drawn from the living animal generally separates on coagulation, the serum being reddish (because it contains haemoglobin) ; but in some cases this is lacking, probably when the haemoglobin has been already excreted from the body. Moreover, according to the investigations of Siedamgrotzky and Hofmeister, the blood is very rich in urea and in extractive substance, the products of the increased muscular metabolism. Lastly, we find here and there under the microscope an immense number of haematoid crystals in the blood.

Besides these more important changes, which are found in every case examined, certain secondary affections of the internal organs must be named. When, for instance, the muscular haemoglobin has reached the arterial system, the body at once begins trying to extract it again from the blood by the apparatus at its disposal. To these belong first of all the spleen, which receives the dregs of the red, blood-corpuscles, and becomes consequently swollen ("spodogenous" tumour of the spleen, according to Ponfick) ; then the liver, which also is usually more or less swollen, and the red medulla of the bones, which is first found infiltrated with sugar and dyed an intense black-red. This swelling and infiltration of the red medulla, which are (though not always) noticed in the larger bones (femur, humerus, radius, tibia) must not be regarded as osteomyelitis. Excretion of the haemoglobin through the kidneys, or haemoglobinuria, only begins then when the amount of free haemo-

globin circulating in the blood exceeds one-sixtieth part of the total haemoglobin of the body. In the latter case a haemoglobinous congestion is produced in the kidneys, to which further changes, and especially acute parenchymatous nephritis, may be added. Finally, hemorrhage occurs occasionally in single organs (spleen, kidneys, etc.), and septicæmia may easily develop in connection with decubital gangrene.

**Symptoms.**—The signs of rheumatic haemoglobinæmia in horses are in general as follows:—In most cases the animals, having stood idly and well fed in their stalls for some days, where the air has been very warm and ventilation bad, on returning to work shortly begin to manifest irregularities of movement. This is usually in a quarter to half an hour after they start, but may be sooner or later. In slighter cases, the impression is gained that they have suddenly been smitten by rheumatism. They seem stiff, or even lame, in the hind limbs especially, or sometimes in only one hind or fore leg. At the same time partial sweating breaks out. In severer cases the horse stagger on their hind legs, have a stiff and insecure gait, the hinder limbs seem difficult to bring forward, the angles of the joints remain more widely open, so that the whole limb looks longer, and the hoofs are dragged on the ground. Then the animal gives way at the pastern, etc., trembles, sweats, can with difficulty stand on its hind legs and, not unfrequently, tumbles on the ground as though struck by apoplexy. When on the ground, it makes frantic attempts to rise, works its legs about, shows signs of difficulty of breathing and of terror, and is occasionally bathed in sweat. It can then only be transported to its stable on a wagon or sleigh.

On *palpation* of the paralysed hinder parts, the muscles are found very hard, tense and painful, especially those of the croup. The skin of this part is higher in temperature and swollen like a board (*oedematous infiltration of the perimysium, subcutis and cutis*). The sensibility of the parts to touch, to pin-pricks or electricity, is often unimpaired, but not rarely is quite suspended. Varying from these conditions, we sometimes notice paralysis of other parts of the body, such as its front portion, or one foreleg, the shoulder muscles, upper neck-muscles, etc.; but this less frequently. According to Bouley, among ten cases, the left hind leg was first affected. Sometimes the tail is held quite stiffly. We have often seen agonised contractions of the muscles of the belly, and the animals have screamed with pain.

Another striking symptom is the haemoglobinuria, which appears in all severer stages of the malady. This was formerly considered a pathological and invariable sign of the disease. But it is evident from pathogenesis, and has been confirmed by much experience, that haemoglobin may be absent from the urine, that is, in slight cases, or in those with abortive course. This shows how unsuitable was the former name of haemoglobinuria for the disease. The urine which contains haemoglobin or methaemoglobin, is noticeable for its dark red, at times ruby-red, or from dirty-brown to an almost inky-black colour. With regard to the reaction of the urine, we must affirm, in opposition to what others have said, that in slighter cases it almost always remains alkaline, and even in severer cases, is not rarely permanently so. Thus, in twenty-five carefully tested cases, five of which ended fatally, and which on the average might all be reckoned severe, we only once found an acid urine (=4 per cent.), which was very rich in phosphates. According to French practitioners, the urine should contain sugar. The specific gravity is in many cases, even severe ones, found to be quite normal. Taking all these facts together, we are brought to the conclusion, that in haemoglobinæmia of horses the urine possesses no diagnostic importance.

There is usually no rise of internal temperature, even in very severe cases, which we affirm in contradiction to what some have stated. Thus, among 25 cases, we found 20 without any such rise; in four the temperature exceeded 102° Fahr., and in only one was above 104° Fahr. Engel also found in four cases hardly any fever. But the pulse, on the contrary, is usually quickened. Thus in the above 25 cases, we found no acceleration in four cases; in 16 the rate exceeded 60 a minute, and in five was above 50. But it not unfrequently rises to 70 or 80 and more, being then hard and jerky. The visible mucous membranes are usually redder and of a dirty colour (signs of blood-dissolution). The bodily temperature is unevenly distributed, the extremities being cold. In consequence of the considerable change in the blood, decubitus often appears when the animals have only lain ill a short time (haunches, edge of zygoma, etc.).

In the digestive organs the diminution, or even suppression, of peristalsis is in very many cases very noticeable. Other changes do not, however, occur, appetite being normal, or even increased. Coincident with the suppression of peristalsis, there is retention of urine in the bladder, but this can usually

be remedied by pressure through the rectum. The difficulty of breathing, which accompanies the course of this disease, has been already mentioned.

The sensorium is perfectly unaffected, and disturbance only occurs when there is complication with nephritis. Thus, in one case we noticed uræmic symptoms, which manifested themselves in much dulness of the sensorium, and in epileptiform spasms. The cerebral disorder and signs resembling staggers, which some have noted, may be thus explained. Such indications are, however, essentially secondary.

**Course and Duration.**—The course is generally acute, and even subacute. If the patient recover, such recovery may take place in an hour or two (very slight, abortive cases), or within a few days, generally in two or three. When this happens, the recovery is often a complete one; but we must not forget that the course of this disease is not seldom marked by periodical increases of violence, and after apparent improvement relapse may occur. For instance, a horse may fall ill several times in a year, or for several successive years on the same day, if kept a few days in its stall. Occasionally the same animal will be ill several times in a few weeks. A simultaneous affection of several horses in the same stable has also been observed. Evidently, then, no immunity is gained by having once passed through the disease, but rather, on the contrary, a predisposition to further attack. The same thing has been noticed in paroxysmal hæmoglobinæmia of man, which has therefore been called "periodical," or "intermittent" hæmoglobinæmia.

In severe cases, recovery often leaves a paralytic condition of the extremities behind. Either a paresis of both sides of the hind quarters remains for weeks, months or even years, so that the hind limbs appear lame, the feet drag and the gait is uncertain; or else a permanent paralysis of the extremities and of certain groups of muscles is developed on one side, with muscular atrophy. Thus, paralysis of the groups of muscles which receive their strength from the nervous tibial and crural nerves has been repeatedly noticed. Excessive atrophy is sometimes observed in the muscles of the buttocks. We also saw a case in which chronic meningitis spinalis, with paresis of the hind legs, of the bladder and of the bowels, occurred as a sequel of hæmoglobinæmia. On dissection we also found, as a very

rare complication, inflammation of the spinal meninges on the cauda equina.

When the sickness ends in death, the paretic conditions grow more and more intense, and pass finally into complete paralysis. The animal grows more restless, the breathing becomes dyspnceic, and decubitus constant. Consciousness grows gradually duller, and death ensues with the appearances either of carbonic acid poisoning, or of paralysis of the heart, or of uremia.

**Prognosis.**—This differs according to the intensity of the symptoms. But it is always well, even in apparently slight cases, to consider the possibility of relapse or periodical recurrences, and to form one's opinion with caution. When the animal is unable to rise and cannot even be lifted by the use of pulleys (complete paralysis), then the forecast is unfavourable, although occasionally horses recover after lying thus for days. The mortality varies much according to race, conditions of weather, individual disposition and the degree of sickness. Sometimes the patients recover one after the other surprisingly well and quickly. At other times one has a whole series of deaths to record. For this reason the mortality estimates vary largely. Bay reckoned that 70 per cent. died out of 368 cases observed, and others have given 60, 50, 40 and even 20 per cent. as the results they noted. In the Prussian army the percentage over four years was from 40 to 50 per cent. In general we may set it from 20 to 40 per cent., but in cases of pronounced paralysis at 50 to 70 per cent.

**Differential Diagnosis.**—Here quite a number of complaints have to be taken into consideration, with which haemoglobinæmia may be, and often is, confounded. The most important are :—

1. *Colic*, particularly the rheumatic colic caused by chill, which, owing to its painfulness, the accompanying suppression of peristalsis and frequent persistent lying down, closely resembles haemoglobinæmia. But the previous history of the case, particularly of long standing in the stall, is a good guide, when combined with examination of the croup-muscles and the urine and with evidence that the animal cannot get up unaided.

2. *Muscular Rheumatism*.—Proof of haemoglobinuria, the patient's previous history, as well as the apoplectiform appearance of paresis or paralysis, assure the diagnosis in all cases. Wide-

spread acute muscular rheumatism without haemoglobinuria is rare in horses.

3. *Thrombosis of the posterior aorta and its branches* (iliac, pelvic and femoral arteries) produces, not rarely, an aspect closely resembling rheumatic haemoglobinæmia and which is, at the first glance, not easy to distinguish. But exploration through the rectum will at once supply the needed information. Moreover, in plugging of the aorta, the severer symptoms vanish very quickly and give place usually to a complete restoration.

4. *Apoplexy of the spinal medulla and also spinal paralysis and paresis of the hind leg* of traumatic or inflammatory origin, may likewise give rise to confusion. But the traumatic mode of development and gradual growth of a meningitis spiralis, as well as absence of any primary foci for emboli with apoplexy, all furnish hints to the observer. But the nature of the urine will be especially significant.

5. *Tetanus*—Mistakes may possibly be caused by the firm, tense condition of the croup-muscles. But on more minute comparison of the other symptoms, differentiation should not be difficult. The development of tetanus is essentially slower, and haemoglobinuria is never present.

6. *Nephritis, Anthrax, Petechial Fever, etc.*, are all easy to distinguish with a little care.

**Therapeutics.**—In the treatment of haemoglobinæmia of horses prophylaxis plays the first part. The disease can be almost certainly prevented by exercising the horses daily, even on holidays, and by feeding them less liberally on such days. They are thereby hardened against any weather, and relapse is especially avoided. This treatment must be aided by rational ventilation and heating of the stable.

When once the disease has broken out, it is first of all necessary to get the horse to its feet and, if possible, to suspend it in a sling, as, when lying down, decubitus and hypostasis of the lungs quickly develop with fatal effect. But we must draw attention to the fact that horses which are unable to support themselves when slung run great risk of quickly choking. In all such cases as, by reason of extensive blood-dissolution, develop great difficulty of breathing or abnormal acceleration of the heart, it is well to bleed the animal freely. This, our opinion, is confirmed by others, but we must remind our readers that the blood is sometimes very thick and flows badly. If, as often happens, paresis of the bladder produces retention

of urine, attempts must be made to empty the urine, either by pressure of the flat hand from the rectum, or by introduction of a catheter. Moreover, in order to assist extraction of the haemoglobin through the kidneys, we add as much water as possible to the drink, and seek to stimulate the action of skin by friction and rubbing in of spirituous and stimulating agents, such as spirits of camphor with oil of turpentine, and that of the bowels by purging and even drastic remedies. Physostigmine is good for the latter purpose, owing to its easy application and certain operation. It should be injected in a 10 per cent. solution (mixed in water), in doses of 1½-2 grains under the skin of the neck or shoulder. Also pilocarpine (3-12 grains) and arecolin (1½ grains) may be used. For the muscular paralysis which remains, massage is recommended, also veratrine (½ grain) and caffeine (8-12 grains); strychnine less so (myopathic paralysis). Reports are not favourable to the use of intra-tracheal iodine injections. Dietetic treatment consists in meal-drinks, bran-mash, carrots, green food or good hay. Richer food, especially oats, should be avoided. The convalescents must be gradually accustomed to exercise.

Utz and Vaeth describe a peculiar sub-acute form of haemoglobinæmia. In this the general symptoms do not appear suddenly, but gradually, so that the animals are not known to be ill until the third or fourth day. Utz also noticed muscular swellings on the masseter, followed by difficulties of mastication (trismus). Friedberger also witnessed marked symptoms of trismus in two cases of haemoglobinæmia. As for the cases of infectious or toxicæmic haemoglobinæmia, they sometimes resemble in aspect mycotic intestinal inflammation or other cases of poisoning, and seem, consequently, to belong rather to the latter category.

#### HæMOGLOBINÆMIA OF CATTLE AND OTHER RUMINANTS.

(*Bloody Urine, Red Water, etc.*).

**Definition.**—This disease, which is described in literature under a great variety of local names, is one of the most obscure in the whole range of veterinary pathology. Both in the opinions and descriptions of authors there is the widest divergence. First of all we must exclude from the current general term of "bloody urine" the enzoötic gastro enteritis in its complication with nephritis hæmorrhagica, as well as with hæmaturia, a symptom referring to nephritis. The latter disease has been separately discussed in our chapter on poisoning. If, then, to the name "bloody urine," in its

narrower sense, we have added that of "haemoglobinæmia," it is done by reason of full examination of existing literature and of our own observations. These demonstrate positively that bloody-urine is not to be regarded as haematuria, but as haemoglobinuria. The haemoglobinæmia of cattle is therefore closely allied to that of horses, as further facts will more strongly confirm. Our conception of the nature of the disease is that bloody urine is in the main a decomposition of the blood induced by infectious or toxic substances (infectious and toxic haemoglobinæmia), and partly, also, a myogenous, rheumatic haemoglobinæmia. Its epidemic occurrence is partially caused by protozoan parasites in the blood-corpuses (piroplasma).

**Occurrence.**—Haemoglobinæmia occurs most frequently among cattle, but also among sheep and goats, and in by far the larger number of cases appears enzootically in pasturing cattle, more rarely and sporadically in the stable. It is a very ancient disease and was known to the Greeks and Romans, is spread over all Europe, but appears to be diminishing with the advance of cultivation. At present it especially attacks herds in Alpine districts and in the Northern hills and plains. In some parts they speak of certain "red-water farms." These are mostly on low-lying land, surrounded by forest and overgrown with alder bushes.

Females and young animals are chiefly affected, also milch-cows; but the males less so. Several observers have noted—and this is important—that cattle born and brought up on the spot show much greater power of resistance than those newly imported. Certain individuals also seem more predisposed. It often happens that the same cow is affected twice a year, viz., in spring and autumn, and even several times during one term of pasturage. It seems to be especially a spring complaint, and afflicts particularly such herds as have passed the winter on short rations and in crowded byres, and been then driven out early in wind and cold to feed on pastures as yet hardly grown. The disease is fixed and stationary on such cold, windy, shaded pastures as are also poor, peaty and marshy. It usually breaks out at the end of the first week or beginning of the second after arrival—even sooner if this be too early—and lasts until about the middle of summer. According to Gerlach, it may break out eight to ten days after the animals have been removed from the place. The complaint is not, however, confined to spring, but has been known to appear in the middle of summer

after cold, wet weather. In the Alps it is said only to occur upon slate rock and not upon chalk formations (Lechner).

**Etiology.**—By the greater number of observers the bloody-urine of cattle is regarded as a result of decomposition of the blood. As for the nature of such decomposition, everything points to haemoglobinæmia as a foundation of the disease, for red blood-corpuscles have never yet been with certainty demonstrated in the urine. Although the essential causes of this haemoglobinæmia are not yet known, yet there is a consensus of opinion that two chief factors—which perhaps act in co-operation—may be looked upon as its proximate sources, viz., infectious-toxic substances on the one hand, which have been absorbed with food and which decompose the red blood-corpuscles; and, on the other hand, the action of cold, producing probably the same results as in haemoglobinæmia of horses.

i. The infectious-toxic influences are traceable to peculiar properties of the soil and a corresponding change in the nature and composition of its vegetation. The facts that the disease is often local and confined to certain pastures, and that bloody-urine (Blutharnen) often breaks out in the byres among cattle fed from hay cut on these pastures, both point to the presence of some infectious agent in the vegetation. The symptoms which we still have to detail also indicate poison, which, being taken in the food, causes, first of all, diarrhoea or haemorrhagic gastro-enteritis and, after absorption into the blood, decomposes the latter. The observations of several authors, who have noted outbreaks of bloody-urine in cattle after eating unsound food, such as putrid turnips or beetroots and putrid leaves of the same, distillery refuse and fodder infested by moulds, point to some, at least, of the cases of haemoglobinæmia being of mycotic character. Finally, opinions are almost unanimous that the disease is particularly confined to certain swampy, boggy meadows and forest tracts, which fact justifies the assumption that it is caused by some malarial miasma traceable to a bacillus.

The greater number of poisonous plants, so-called "bloody-urine plants," produce less haemoglobinuria than haematuria. Among these are those named in connection with enzootic intestinal inflammation, viz., fir-tree sprouts, ranunculus, broom, water-pepper, mercury and the various kinds of euphorbia and hemlock. It is doubtful whether the sour or half grasses (*carex*, *scirpus*, *juncus*) blamed by Spinola, have any connection with bloody-urine. On the other hand, the alder (*alnus alpina*,

*viridis, incana and alnobotula*), is so far connected therewith, that pastures overgrown with alder bushes appear to be peculiarly injurious.

2. Many circumstances point to the co-operation of a rheumatic cause, as in the hæmoglobinæmia of horses (rheumatic or myogenous hæmoglobinæmia). First, the complaint occurs most frequently on transfer from the byres to cold, wet and stormy spring pasturage, and does not break out on transfer at later date. It may also be produced by serving very cold water in the byre, or by drinking from mountain streams in the open. According to Röll, it occurs in Galicia mostly in cold, mountainous districts. Dotter considers bloody-urine essentially a rheumatic ailment; Utz also blames cold especially as a prime cause, and has seen the complaint break out in the byres during a very cold season. Krug noticed an outbreak among draught-oxen on the same day that the disease appeared among horses. Hink describes what he calls "pasture-red" as being common in the Black Forest. It is much like black disury of horses, and attacks especially young cattle the first time they are turned out to pasture. It is caused by cold and by greedily devouring wet, chilly fodder, combined with unaccustomed exertion. Finally, Saur saw a well-nourished milch-cow which undoubtedly sickened of general rheumatism, with which hæmoglobinuria was complicated. These factors, combined with a similarity of many clinical symptoms in both diseases (stiffness of hind limbs, sensibility to touch in the renal region), indicate, in our opinion, a relationship between the hæmoglobinæmia of cattle and that of horses.

**Hæmatococcus.**—The etiology of hæmoglobinæmia of horses has been of late years much advanced by the researches of Babes, Starcovici and others. Babes reports upon a plague among cattle in the swampy Danube districts of Roumania, in which 30,000 to 50,000 oxen perish yearly. Cows sicken more rarely and mostly recover, calves escape altogether. Its symptoms are in general the same as those of hæmoglobinæmia of cattle. But whether it be the same complaint as the bloody-urine of Germany cannot yet be positively stated. But the most important symptom is the presence of hæmoglobin in the urine. Babes discovered a diplococcus to be the cause of the plague, which he found present in the blood, i.e., in the interior of the red blood-corpuscles, in the kidneys, stomach, etc. This hæmatococcus produces hæmoglobinæmia, says Babes, by decomposition of the red-blood-corpuscles. The organism can be best shown bacteriologically by first staining it with Löffler's methyl blue solution and subsequently with concentrated methyl blue solution, with final treatment in aniline oil and xylol. Transfusion of the blood of sick animals to healthy ones produced no effect. Sheep, pigs, fowls, doves and guinea-pigs all resisted inoculation.

Rabbits, on the other hand, developed inflammation of the bowels, peritonitis, pleuritis and pericarditis after being inoculated. Piana has described a similar local plague in the mountains of Brescia, which has wrought yearly damage there to the amount of £20,000. He found small cocci in the sick animals' blood, and was able to transmit the disease to sound cattle by intraperitoneal injection of infected blood. After sustaining the effects of two or three inoculations, the animals are said to become immune.

According to Starcovici, the epidemic haemoglobinuria of the Roumanian cattle (Babes), the Texas fever of cattle (Smith), and the so-called Carceag of Roumanian sheep (Babes), are all caused by the same protozoön, i.e., by varieties of the same. Compare Texas fever and Carceag (Vol. I., pp. 415 and 416).

(NOTE.—Most of the so-called red water is probably due to piroplasmosis, the micro-organisms being conveyed by ticks.)

**Symptoms.**—In describing the symptoms of bloody-urine recent authors have largely mixed those of haematuria renalis with the signs of genuine haemoglobinuria. We have, therefore, had to confine ourselves mainly to the descriptions of older authors (Spinola, Stockfleth, Gerlach), from whose statements some modern publications deviate considerably. These older authorities unanimously agree that the most important symptoms are—stiffness of the hind legs, increased sensibility to touch in the renal region, and the passage of urine more or less red in colour, but containing no blood-corpuscles. The disease usually begins with high fever ( $104^{\circ}$ - $105.8^{\circ}$  Fahr.), to which diarrhoea is added, that may even be bloody. Appetite, which at first is suppressed, is later normal, or only slightly diminished, and only a general lassitude, with slight rise of temperature, is to be noticed. Sometimes within six to twelve hours the urine, at first pale, shows a more or less dark to brown and black-red colour, or even looks like tar. It also contains much albumen or haemoglobin, in consequence of which it forms on being passed, and, when boiled, stiffens to a dark brown, pulpy or gelatinous consistence. It readily forms a muddy sediment, and in sheep, dyes the wool red all about the place of discharge. During the first three to five days the reaction, according to Reuter, is acid, but later becomes alkaline (note the ammoniacal smell of the urine). It is an interesting fact that the disease may occur without any red colour of the urine (Stockfleth), whereby its similarity to haemoglobinæmia of horses is increased. Urine as just described is passed at first in larger quantity, which then often greatly diminishes, being passed with much evidence of pain (strangury). Upon the diarrhoea there mostly follows on the third day an obstinate constipation, during which tympany

is not seldom observed. The internal bodily temperature rises to 106° Fahr. or even higher.

To these symptoms motor disturbances in the hind legs are soon added, viz., stiff, constrained and clumsy gait, awkwardness and even pain in lying down or rising, arching of the back, together with an intense sensibility of loins and croup which is sometimes quite striking; also slight twitchings or even feeble spasms in the hinder parts, while spiral movements on the perineum along the urethra have been noticed during micturition. Finally, the weakness on the hinder parts grows more and more marked, until at last the animal can no longer stand. In the last stage of the disease sometimes oedematous swellings appear on the head, neck, point of the chest, etc. The mucous membranes are either reddened, or else, during later stages, dyed a dirty or citron yellow. The milk itself occasionally assumes a yellowish-red colour and the icterus becomes general (Gerlach). Meanwhile the pulse grows more rapid but feebler, exhaustion prevails, the temperature sinks all over the body, and death ensues with a general collapse of strength. The urine may for some time prior to death have again become clear.

**Course.**—This is acute; more rarely peracute, when the animals may perish apoplectically in a few hours (Reuter). If the causes be quickly removed or only operate feebly, the course is usually quite benign, but is generally very variable. Its duration averages 10 to 14 days, and recovery occurs in the majority of cases. Stockfleth reports 23 deaths among 79 observed; Krabbe only 38 among 330; and Rudovsky 6 out of 50. But recovery does not protect from relapse. Reuter saw cows affected by bloody-urine thrice during one period of pasturage. The same author says that in some communes the disease may be so destructive as to carry off 30 to 40 per cent. of the total cattle.

**Post-mortem Lesions.**—These are usually not very important. The carcases are mostly quite anaemic, the blood very fluid, from cherry-red to inky-black in colour. The blood-serum separated by coagulation is at times red (contains haemoglobin). General icterus is often present. There are no organic changes, except perhaps an acute intestinal catarrh, or hemorrhagic enteritis. The kidneys especially are generally normal, but the spleen is occasionally enlarged. According to Spinola

and Hink, the muscles are slack and pale. Reuter noticed them of faded colour, as though boiled; and in several places, particularly about the neck, shoulders and hinder thighs, saw more or less diffuse, bloody-serous or gelatinous infiltrations. (The similarity of this condition to that in rheumatic haemoglobinæmia of horses is evident.) Stockfleth ascribes the cause of death partly to extreme anaemia, which is most frequent, and partly to secondary pneumonia.

**Differential Diagnosis.**—Among diseases which may be confounded with haemoglobinæmia of cattle are, first, various affections of the kidneys which are accompanied by haematuria, especially enzootic inflammation of the bowels. But they are not hard to distinguish, if the urine be duly examined. Anthrax, which also is accompanied by haematuria, and perhaps with haemoglobinuria as well, may cause mistake. In this instance the whole process of the disease, combined with examination of the blood, must furnish the indications needed. Finally, the dark, brownish-yellow urine in liver complaints or icterus may be thought to contain haemoglobin. Here the question lies in proving the presence of biliary pigment.

**Therapeutics.**—Prophylaxis is here of the utmost importance. This consists in not driving out the animals too early in spring to pasture, in not overcrowding the byres, and in feeding with raw fodder a little while before taking the cattle out. Then the ground should be improved by drainage and cultivation; and, finally, a native-born, more resistant breed of cattle should be raised. When the disease appears, cease pasturing at once and take the herd back to cover. The disease itself is hard to combat. First, according to the symptoms, we must treat the constipation with laxatives, and later must attack the diarrhoea by styptic remedies (sulphate of iron, 4-6 drams; alum, 4-6 drams; tannin, 4-6 drams; sugar of lead, 2-4 scr.; opium, 2½-5 drams). Some prescribe subcutaneous or internal use of santonin. Blood-letting in prolonged cases or with excessive anaemia is objectionable. A transfusion of blood may be attempted in severer cases (Stockfleth).

**Haemoglobinæmia in other Animals.**—Besides horses and cattle and smaller ruminants, especially goats, this disease has been noticed in other animals, such as mules, dogs (in consequence of chill after poisoning or during distemper), in zebras and also in pigs. Utz

found dark-coloured urine, rich in albumen, and also, along with other symptoms, a yellow colour in the fat, in a pig which had been transported about five miles during a cold of  $13-16.6^{\circ}$  below zero, Fahr. Chill seems therefore to be a factor in producing haemoglobinuria in the other domestic animals. Respecting haemoglobinuria caused by blood parasites in cattle (Texas fever) and in sheep (Carceag), consult the chapter on Infectious Diseases (Vol. I., pp. 415 and 416).

## CHAPTER VIII.

### DISEASES OF THE SEXUAL ORGANS.

#### PUERPERAL FEVER.

(*Milk-fever, Calf-fever, Septicæmia and Eclampsia puerperalis.*)

**Ancient and Modern Views on the Nature of Puerperal Fever.**—The acute diseases of the mother animal after the birth of her young have always engaged the attention of veterinary surgeons, especially as affecting cattle. Of this fact the numerous names under which such diseases are known afford abundant evidence (calf-fever, milk-fever, puerperal fever, paresis of parturition, septicæmia, eclampsia puerperalis and many others). But the symptoms noted have been, from the very first, by no means uniform. So varied are they, in fact, that Hering remarks : "Almost every description seems to refer to a different disease." No wonder, then, that opinions have varied widely as to the origin and connection of these symptoms ; some authors, and they the majority, have spoken of various forms of puerperal fever—the inflammatory-septicæmic and the nervous-paralytic—while others only recognise one form, viz., that which is accompanied by paralytic symptoms, and have strictly separated all others, especially that called "inflammatory," and classed them among diseases of the uterus. In later years, following the lead of Franck, authors have pretty generally ceased to speak of two *forms* of calf-fever, but have divided this into two completely separate diseases, according to their nature and their anatomical and clinical aspects.

I. *Puerperal fever*, or *Septicæmia puerperalis*, the inflammatory calf-fever of older authors, which has also been termed phlegmonous or diphtheritic inflammation of the uterus, or gangrene of the uterus. This consists in an intense inflam-

mation of the vagina and uterus with consequent general sepsis, caused by reception of septic matter.

2. *Eclampsia puerperalis*, corresponding to the nervous or paralytic form of the older authors, and, not very happily, named "calf-fever" or "milk-fever." According to Franck, this consists in congestion of the brain produced by too rapid contraction of the uterus, with subsequent oedema and anaemia of the brain, manifesting itself in loss of consciousness and general paralytic phenomena, or less frequently in spasms. This theory has been opposed by Schmidt-Mülheim. In his opinion, paralytic calf-fever develops as a sequel to peculiar decompositions of the albuminous lochial fluid in the cavity of the womb, which has been shut off from the air by too rapid contraction of the uterus, such process being different from putrefaction. In this manner a poisonous agent (very similar to that of meat or sausage poisoning) is generated, which, on being absorbed into the circulation, produces symptoms of poisoning quite resembling those of meat or sausage poisoning in man. Such are, first of all, paralytic weakness of the involuntary muscles, of the extremities, trunk, tongue, pharynx, velum palati, larynx and upper eyelid (*ptosis*) ; also paralysis of the smooth muscular apparatus of the bowels and vessels, accompanied by an absence of all more pronounced changes.

**Criticism of Franck's Theory of the Pathogenesis of Paralytic Calf-fever.**—The division, first effected by Franck, of the conception of "Puerperal fever" into two quite distinct groups of disease, viz : the septicæmic and the paralytic, must be regarded as an important advance in the science of puerperal disorders. In this division we entirely agree. But, on the other hand, we must declare Franck's theory of the production of puerperal paralysis to be scientifically untenable. This very complicated disease cannot be explained by mere disturbances of circulation. Indeed, the name "eclampsia," chosen by Franck, is wrong. Under eclampsia we understand a form of spasm which shows itself in convulsions, and, indeed, in pronounced epileptiform spasms, and may to some extent be regarded as an acute epilepsy. Such an eclampsia occurs among our domestic animals, e.g., with suckling bitches after parturition, and forms, clinically considered, the exact opposite of paralytic calf-fever in cattle. (Compare the paragraph on eclampsia in dogs in our last chapter on Nervous Diseases, Vol. II., Chap. XII.)

Also in man, eclampsia parturientum manifests itself in spasmodic attacks, lasting minutes, with intervening pauses of calm, which arise in immediate connection with the act of birth, and generally in first confinements—sometimes also as the outcome of an uræmic condition (nephritis). In contrast to this, paralytic calf-fever represents a paralysis which often lasts very long, and only appears some days after delivery and, as a rule, in those which have previously borne several young.

But, apart from this mistaken nomenclature, paralytic calf-fever cannot be explained according to the theory given by Franck. Anæmia of the brain, with œdema of the same, show essentially other symptoms, and a much more fatal course; moreover, they cannot always be with certainty demonstrated in calf-fever. Further, the frequent, sudden disappearance of all signs of illness in this disease cannot be explained by the presence of œdema of the brain. To these comes the fact that in rare cases the disease is observed before parturition, which is quite inexplicable by Franck's theory. But above all, the latter fails to give any plausible explanation of the well-known fact, that the disease does not usually appear until several days after the birth, whereas such disturbances of circulation as occur must be immediately connected with the act of parturition.

On these grounds it can easily be understood, that we have sought some other explanation of paralytic calf-fever. This we found in the theory of Schmidt-Mülheim. This theory we fully accepted in our first edition, and can to-day only confirm that acceptance, the appropriate appreciation of which demands, however, a certain toxicological knowledge. Our opinion has, in the meantime, been only strengthened by the advance of science (Thomassen, Baas, Stubbe). The presumption of an auto-poisoning by ptomaines, or leucomaines as the cause of paralytic calf-fever seems very reasonable, especially when we consider the extraordinary resemblance between the aspect of this disease and that of ptomaine poisoning, as revealed in literature, or experimentally produced. In sausage poisoning (botulismus, allantiasis; see the chap. on mycotic gastro-enteritis, Vol. II. p. 169), quite characteristic paralyses of certain muscular groups also occur, just as in paralytic calf-fever—for instance, of the upper pharynx and the eye (paralysed deglutition, ptosis). These are caused by a recently discovered ptomaine present in the poisonous food, viz.: ptomatoatropin. One of the commonest bases of putre-

faction is ptomatocurarin, also a ptomaine, which in its operation agrees entirely with curare, and likewise produces paralysis of certain muscular groups. Such ptomaines find most favourable conditions for their development in the womb. The circumstances that paralytic calf-fever generally sets in a few days after parturition, is explained by the fact that an interval of some days is needed for the ptomaines to develop in the uterus. This removes also the objection that ptomaine poisonings develop more slowly than paresis of parturition. The period between the birth and the first appearance of the malady represents then, in some degree, the incubation stage of paresis of parturition. The rapid disappearance of all paralytic signs occasionally noted, may be naturally explained by rapid removal of the poison from the body. Those cases, finally, in which such paralytic symptoms show themselves prior to birth, may find their explanation in the fact that ptomaines have already formed in the womb before parturition, when the opening stage of delivery is of long duration, that is, when a partial opening of the womb has preceded the act of birth (differentiation between the opening and expulsion stages in parturition). But on Franck's theory this circumstance cannot be explained at all.

#### I.—SEPTICÆMIA PUERPERALIS.

(*Septic or Pyæmic Febris Puerperalis.*)

**Occurrence.**—Septicæmia puerperalis occurs in all our domestic animals, most frequently among carnivora, but not seldom in cattle. The essential cause is to be sought in the absorption of septic matter, arising either from putrid after-birth, or dead foetus (self-infection), or communicated from without (infection by the obstetrician, by the instruments used, forceps, etc., or by neighbouring animals suffering from puerperal fever, abortion, etc.). Entrance is provided by wounds on the mucous membrane, caused by clumsy assistants, hard parturition, change of position by the young, the operation of embryotomy, or too early and forcible removal of the after-birth. The disease does not usually show itself immediately after birth, but a few days later, in cows mostly on the third day.

**Post-mortem Lesions.**—Except in the rare cases which run a very acute course, and in which no inflammation

of the uterus takes place, we find ulcers—so-called puerperal ulcers—on the mucous membranes of the vagina, cervix uteri and uterus upon the sites of the original wounds. These are covered with a discoloured, necrotic or diphtheritic deposit, which has sometimes become detached and been replaced by granulations. Around these ulcers the mucous membranes are oedematosely swollen. Here and there the swelling attacks the external genital parts, and extends even to the inner sides of the thighs and belly. The remaining mucous membrane of vagina and uterus are also in a state of phlegmonous inflammation, which, in dogs, frequently develops into diphtheritic scurifness (*kolpitis* and *endometritis phlegmonosa crouposa* and *diphtheritica*). In the latter case, it is of a dirty brown, or greenish-black colour, permeated by ulcers and covered with ichorous, foetid matter. In some spots it is quite changed into a greasy, necrotic mass, on removal of which the muscles of the uterus are laid bare.

The parenchyma of the uterus is itself oedematous and ulcerated. The lymphatic vessels are charged with pus, the veins with thrombi. Under the microscope, the lymphatic spaces of the muscular layers are seen to be thickly filled with colonies of micrococci. The uterus, moreover, is badly contracted, its walls are friable, flabby, and their muscular fibres in a condition of opaque swelling or fatty degeneration. Sometimes there are abscesses among the muscles (*metritis phlegmonosa*). The connective tissue around the uterus is likewise oedematous, swollen and infiltrated with pus, and traversed by abscesses (*parametritis phlegmonosa*). Further, the peritoneum surrounding the uterus, and covering the pelvis, and finally the whole peritoneum are in a state of serous, purulent, ichorous inflammation (*perimetritis*, *pelvo-peritonitis*, *peritonitis diffusa*).

If the condition be sufficiently advanced, we obtain now the aspect of septicæmia and pyæmia. We find purulent pleuritis, pericarditis, ulcerative endocarditis, meningitis, arthritis and hæmorrhagic gastro-enteritis. Furthermore we perceive metastatic inflammation of lungs and kidneys, abscesses in the mediastinum, in the subcutaneous and inter-muscular tissue, and in the soft parts enclosed by the claws; swelling and degeneration of spleen, liver, myocardium, mesenteric and lymphatic glands especially; formation of thrombi in the vessels of lungs, liver and kidneys; ecchymosis in all organs, particularly under the endocardium; greasy, badly coagulated, tar-like blood,

which infiltrates the tissues ; decomposed red blood-corpuses, and a rapid decomposition of the body, with much generation of gas.

**Symptoms.**—Except in the rare cases of rapidly developed general sepsis with very acute course, the disease usually begins with signs of inflammation in the genital passages. First, the mucous membrane of the vagina and vulva is found to be reddened, swollen, higher in temperature and covered with diphtheritic ulcers and ichorous secretion. Occasionally the phlegmonous swelling of the vulva extends to the inner sides of the thighs. On palpation from the right abdominal wall, or from the rectum, the uterus is found to be swollen and painful. Ulcers are frequently found grouped especially about the urethral orifice. Corresponding to these inflammatory affections of the vagina, uterus and urethra are the signs of pain manifested by the animal. It grows restless, wriggles its tail, arches its back, looks backwards, shuffles about, often lies down, contracts its abdominal muscles, presses and strains upon the uterus as though in extreme labour pains, as a result of which a foul-smelling, discoloured, ichorous-looking fluid is discharged, finally it often attempts to make water, but can only pass a few drops with much pain and effort.

At the same time a great rise of temperature is observed, accompanied by shiverings. The rectum will show as much as  $104^{\circ}$  to  $107.6^{\circ}$  Fahr. ; and the pulse, 80, 100 and 120 beats per minute, but the latter becomes small, thread-like, and often also imperceptible. The extremities, such as feet, horns, ears, and even the dry muzzle feel alternately cool and then very hot. If the animal lie long, decubitus quickly sets in. Along with these signs, there is complete cessation of both eating and drinking, and also of milk-secretion, accompanied by constipation and slight tympany. The sensorium is excessively dulled, all movement becomes sluggish and uncertain, feebleness rapidly increases, so that the animals cannot stand. At this stage the disease much resembles the aspect of paresis of parturition, but may be distinguished therefrom by the very high temperature, the inflammatory local changes and the lack of the characteristic muscular paralysis in the eye, the pharynx and the larynx.

The course of the disease may vary in three directions. Either death occurs with increase of the above-named symptoms, sometimes being immediately preceded by diarrhoea,

the end in such cases coming in three or four days, or at most in six (more rarely it is apoplectiform, and follows in one or two days); or complete recovery may be made in eight to fourteen days, with abatement of the inflammatory signs, fever, etc.; or, finally, a chronic condition of illness may develop characterized by a chronic, wearisome illness, with great emaciation, hectic fever attacks, extreme cachexia and final exhaustion; further, chronic endometritis complicated with persistent discharges (*fluor albus*); the mouth of the womb becomes closed, resulting in complete sterility; pyæmic metastasis of the lungs, joints and other organs sets in, as well as chronic inflammation of the kidneys, with its consequent phenomena. The metastatic inflammations just named give the impression of a recurrence of the disease, and may occur several times in succession. Finally, if this condition last long, haemorrhagic diathesis may develop, with haemorrhage in the kidneys (*haematuria*), in the lungs (bleeding at the nose), and in the bowels. We have, in fact, seen distinct petechiae in great numbers on the mucous membrane of the nasal cavity, as in the petechial fever of horses.

The mortality in septicæmia puerperalis amounts to from 50 to 70 per cent.

**Symptoms of Puerperal Fever in Swine.**—These have more resemblance to the paralytic form of puerperal fever in cattle than to septicæmia puerperalis. The disease is marked by a much slighter and less dangerous course, and is commonest among half-English races and those in well-fed condition. Like the paresis of parturition in cattle, it follows chiefly after easy births and after the foetal membranes have been passed, usually three days after birth. According to Herz, the following are the several symptoms:—The animals show diminished appetite and milk secretion, grunt slightly while lying, have an unsteady gait, lie much, stagger and reel, and, at last, avoid every movement of limbs or head, giving the impression of paralysis; take no notice of their surroundings, wear a dull, mournful aspect, keep their eyelids half-closed and sometimes make rotary movements with the bulbus. The bodily temperature is unevenly distributed, that of the interior being  $104\text{--}106.7^{\circ}$  Fahr.), the pulse rises to 140 a minute, the bristles are erected, the skin is hard and dry and somewhat reddened behind the auricles and on the thighs. At intervals a quivering of some groups of muscles is noticed. The vulva and mucous membrane of the vagina are dark red, but only slightly swollen. From the vagina a small quantity of viscid, whitish mucus is exuded. The breathing varies from 20 to 40 per minute and is accompanied by slight groaning. Peristaltic movement is suppressed, and constipation present. The course is acute; recovery follows pretty regularly in one or two days, more rarely in five days. Occasionally weakness in the loins and interstitial mastitis are left behind.

**Symptoms of Puerperal Fever in Sheep and Goats.**—The animals are dull, refuse food, hang their heads, develop an expanded and sensitive belly, arch their backs, and show persistent straining and colic symptoms, as in labour pains. The visible mucous membranes are reddened, temperature rises to  $105.0^{\circ}$  Fahr.; and later a gradual and extreme weakness sets in, with loss of consciousness. The external genitals are more or less swollen, and upon the mucous membranes dark-red streaks and ulcers are seen. The duration of this disease, which is evidently septic, varies from a few hours to, at most, one day. Generally all the animals affected are lost. It is possible that some of the diseases designated as "gangrene of the uterus" in sheep belong to puerperal fever.

**Symptoms of Puerperal Fever in Dogs.**—These are those of a septic metritis; swelling of the genitals, bad-smelling and discoloured discharge from the vagina, very high fever,  $105.8^{\circ}$  Fahr. and more, refusal of food, vomiting, persistent lying, sleepy and comatose condition. The end is mostly fatal within 12 to 24 hours, with great fall of bodily temperature, or else after 12 hours improvement begins. Chronic endometritis is often left behind, as with cattle.

**Symptoms of Puerperal Fever in Horses.**—With the horse also the phenomena of this complaint belong to the realm of septicæmia and consist mainly in higher temperature, quick but weak and irregular pulse, unevenly distributed warmth of body, shiverings, loss of appetite suppressed peristalsis, colic, great stupor, as well as local changes in the genitals. The course is very acute. Koninski states that he saw a case of paresis of parturition in a horse.

**Therapeutics.**—The treatment of septicæmia puerperalis consists mainly in disinfection of uterus and vagina. The aforetime custom of irrigating with carbolic acid (1 to 5 per cent.) may with advantage be replaced by a 1 to 2 per cent. solution of creolin. The latter possesses the advantage of being non-poisonous, and is certainly more effective, as it removes the bad smell at once. It is therefore highly commended by very many practitioners. Lysol is also recommended for rinsing out the uterus. Infusions of sublimate (one per 1,000) are only suitable for the carnivora; for the herbivora, and especially for cattle, it is much too poisonous, and causes great straining—although others have maintained the contrary. Ergot, or fluid extract of hydrastis, may at the same time be administered internally to produce better contraction of the uterus, and thereby lessen the risk of its mucous membrane absorbing septic matter. Gentle purgatives are advisable to assist the excretion of infectious matter through the intestinal canal. The activity of the skin may also be stimulated by friction. Finally, the high temperature and signs of weakness may be symptomatically treated with pyretics and

stimulants. As a prophylactic measure, the strictest cleanliness (asepsis) must be required from the obstetrician (hands and instruments). It is also advisable to isolate highly pregnant animals from others in less advanced condition, and from such as suffer from abortion, chronic discharge from the vagina, or ulcerous skin affections; avoid also any chance transmission of the disease by attendants, implements, food, drink or sponges, and disinfect beforehand the byre with creolin, sublimate, carbolic acid, chloride of lime and other means. Finally, secure good ventilation.

#### II.—PARESIS OF PARTURITION (THE TOXIC OR PARALYTIC FORM OF PUERPERAL FEVER).

**Occurrence.**—Paresis of parturition occurs especially among cows. Evidently the lack of a placenta materna, that is, of a natural wound of the uterus after parturition, has here a determining influence, as the mucous membrane, which is in general intact, absorbs only dissolved toxic matter. It is the same with the uterus of goats, which also suffer not unfrequently from this disease. Yet it occurs likewise in swine. Among cattle, it is evident that milch kine, as well as fat, full-blooded, well-nourished animals of keen appetite, which have been richly fed and kept in their stalls before calving, possess a certain pre-disposition to the disease. This is confirmed by the fact that in districts poor in milk it is of rare occurrence, whereas, on dairy farms, and especially among Dutch and marsh cattle, it causes the most serious losses. Some maintain that certain races inherit such a predisposition. In districts, for instance, where the complaint was unknown, it appeared immediately on the introduction of foreign breeds. Tapken's observations prove that it occurs not unfrequently among pasturing cattle. It is remarkable that paresis appears mostly after easy and rapid deliveries, for which reason cows seldom suffer when bearing their first calf, because the process is then more difficult, and the uterus contracts more strongly than in subsequent births. Generally cows are thus afflicted at the third to fifth birth (Haycock—"Statistics of the Bavarian Veterinary Surgeons"). It has also been established that most cases occur in great summer heat, or during rapid changes of weather. Whether cold has anything to do with it, cannot be decided. The disease usually sets in from 24 to 48 hours after birth, the maximum interval being three days; only very

rarely during deliverance. In isolated cases it has been observed before parturition, but never before the secretion of milk has begun. If, however, we consider that in every birth we must distinguish between the first and second periods of parturition, *i.e.*, between that of opening and that of expulsion, and that often a long interval of time intervenes, we may allow the supposition that those cases apparently noticed before birth really occurred prior to the period of expulsion, but probably after that of opening. In one case of paresis of parturition, which set in 40 hours before the birth, Albrecht, it is true, noticed that the os uteri was completely closed until just prior to parturition.

**Post-mortem Lesions.**—These offer little or nothing that is characteristic. The uterus is usually found strongly contracted and without any injury or inflammatory change. The only thing noticeable is an unequal distribution of blood in the body, in that the viscera of the belly appear very fully charged, as compared with other organs. In many cases the brain shows nothing abnormal; sometimes the meninges are very hyperæmic and traversed by haemorrhages. Hyperæmia of the medulla oblongata has been demonstrated, as well as internal and external hydrocephalus with transuded matter resembling broth (sarcohydrocele). The brain is often found to be very anaemic, flabby and collapsed, and its cortex stained a dirty yellow. In other cases these changes are absent. The cavities of mouth, nose and pharynx are not seldom found quite stuffed with food (paralysis of the swallowing apparatus). The drying-up of the third stomach, formerly regarded as characteristic, occurs in most disorders with prolonged suppression of food-consumption, and is therefore something quite secondary. In all the other organs nothing unusual is to be noticed.

**Symptoms.**—The ailment mostly begins with signs of slight restlessness; the animals shuffle about, strain to evacuate and strike their feet up against their body. More rarely pronounced signs of brain irritation first show themselves; the animal seems excited, bellows and rears up. Still more rarely we perceive epileptiform spasms, twitchings in the muscles of the face, grinding of the teeth and spasmoid twisting of the neck. Some observers have remarked a considerable, hard swelling of the udder at the outset of the complaint.

After these initial stages, which are often overlooked and may, indeed, not be present, the essentially characteristic signs of depression and paralysis appear. First the animal betrays a certain weakness in the use of its extremities, especially the posterior ones, staggers when walking, seems dull and powerless, sometimes falls and struggles to rise again, generally in vain. This paresis soon extends from the hind quarters to the whole body, muscular weakness obtains complete mastery. The animal can no longer stand, but lies helplessly on the ground, its legs half doubled up, or, in extreme feebleness, with all four stretched out.

It is usually in this condition that a medical man first sees it. The brute lies in complete apathy and somnolence, its head held sideways, and the neck so bent that it rests on the side of its breast. If we attempt to lift the head, it falls back powerless into its old position. The upper eyelid droops over the eyeball (*ptosis*), the eyes are shut, and the animal appears to be asleep. On raising the eyelid, the cornea is found to be strangely dull and glazed, as in a dying animal, dry also and insensible, even crinkled and fissured. The pupil is amaurotic and enlarged, secreted tears are collected under the eyelid, the eye-ball is considerably sunken. The tongue hangs loosely from the mouth, owing to paralysis of its muscles, and saliva flows out in long strings, unless its secretion be suppressed, because it can no longer be swallowed. When swallowing fluids, one hears a strange, clucking, gurgling sound all along the throat. Breathing is performed with snoring, groaning, rattling and even whistling noises, accompanied by much distention of the nostrils.

On examining the digestive organs we find obstinate and continuous constipation, caused by paresis of the involuntary muscles of the bowels. This intestinal paralysis generally resists the most powerful purgatives, even *physostigmine*. It is soon followed by tympany and suppression of all intestinal sounds. This is accompanied by cessation of urination, caused by paresis of the muscles of the bladder. The urine contains albumen and also sugar, according to Mocard, even as much as 40 grammes per litre ( $1\frac{1}{2}$  oz. in  $1\frac{1}{2}$  pints). Milk-secretion is likewise suspended, and the udder is relaxed and limp.

With regard to the circulation, we are first struck by the unequal distribution of bodily temperature, the feet, horns and ears especially feeling icy-cold. The pulse is feeble and often imperceptible; the heart-beats are always quickened,

numbering 50 to 70 and later 120 and more per minute. Reports differ as to the internal temperature. Most observers, Franck among them, mention as a characteristically distinguishing sign, in contrast to the septic form of puerperal fever, a steady sinking of bodily temperature to 95° Fahr. and below. This fall, however, does not correspond with the first stage of the disease, but with the second (comatose). During the earlier days of the malady we have seldom missed a low to medium fever 102.2° - 104° Fahr. Engel-Weingarten also noted in eight carefully observed cases of genuine paralytic calf-fever, that in seven instances there was a temperature of 102.2° Fahr. and one of 104° Fahr. This temperature fell in three cases on the first day, in three upon the second and in one upon the fourth day of illness. The conditions of temperature are therefore the same in parturition paresis as in other maladies caused by poisoning. High degrees of fever do not occur in paresis of parturition. If, in any case, the internal temperature rise above 104°-105.8° Fahr., then we must face the possibility of complication with sepsis.

**Course.**—The duration of this disease is somewhat brief. Its course is very acute, and the result may be decided in 12 to 18 hours. In cases of recovery a very noticeable change for the better mostly shows after two or three days. The animals begin to stir, stand up again, appetite and milk-secretion are both renewed as well as the evacuation of dung and urine, bodily temperature becomes more equal, the pulse stronger, and the fallen temperature once more rises. Complete cure usually follows in two to five days from the beginning of illness. Only rarely does a permanent paresis of the hind legs, or a monoplegic paralysis of single extremities remain behind. If, on the contrary, the result be unfavourable, the paralysis passes gradually to the muscles of the heart and to the brain, when death follows with general muscular spasms and restlessness, not unfrequently after sudden diarrhoea. Time, one to three days. Apoplectiform death is sometimes witnessed after the malady has only run a very short time. Owing to the paralysis of the upper pharynx and consequent swallowing the wrong way when administering drugs, the complaint is often complicated with pneumonia due to foreign bodies, from which death ensues sooner or later, mostly in four to six days.

**Prognosis.**—The rate of mortality in the toxic, paralytic

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form of puerperal fever averages from 40 to 50 per cent., but varies, some returns being as low as 25 to 50 per cent. But of those which recover we must reckon 10 per cent. of deaths following upon pneumonia due to foreign bodies. The animals not seldom sicken of parturition paresis.

(NOTE.—By Schmidt's treatment mortality is reduced to 5 to 10 per cent.)

**Differential Diagnosis.**—As milk fever arises in direct connection with the act of delivery, it can only be confounded with a few complaints occurring in the same connection. In this manner the so-called "paralysis," before and after parturition, becomes especially important, as during the course of puerperal fever it may occur simultaneously. This partial paralysis consists in a weakness of the back which appears several weeks before the birth, which cannot be directly removed, but generally disappears on the conclusion of delivery. Or else transient paralytic signs develop in the hind legs after the birth, in consequence of stretching or contusion of the sacral plexus and other nerves, which, however, soon pass away. In both cases the general well-being of the animal is in no way disturbed. With the symptoms of brain-depression which appear in the later course of septicæmia-puerperalis, and which generally precede its fatal end, paresis of parturition cannot well be confounded.

**Therapeutics.**—Prophylaxis is of the utmost importance in paresis of parturition. It should, if possible, include daily exercise of the pregnant animal and decrease of her fodder ration. A gentle laxative (Glauber's salt) is also to be recommended immediately before delivery. Care must be taken to ensure good ventilation and temperate warmth; cold should be prevented. It is best to bring the animal into a separate byre or stable. Moreover, in stables where this disease has frequently caused loss it is advisable to wash out the uterus with a  $\frac{1}{2}$  to 1 per cent. solution of creolin, as a precautionary measure to prevent development of toxic matter. Trasbôt considers that blood-letting, some four or five days before calving, is an effective prophylactic.

When once the disease has appeared, the treatment should aim especially at combating its most serious symptoms, causal treatment being no longer possible. But we must first give warning that the internal administration of drugs is dangerous.

It must, therefore, be strictly limited and carried out with the utmost care, to prevent swallowing the wrong way, with its consequent pneumonia due to foreign bodies. Whenever it can be done, subcutaneous application is preferable. The symptoms to be combated are above all paralysis of the voluntary muscles, as well as of the involuntary ones of the intestines and bladder; likewise, the suppression of brain activity.

1. For the paralytic symptoms and general weakness the use of stimulants is commended, and by preference such as can be applied subcutaneously. Here, first of all comes veratrin ( $1\frac{1}{2}$  to 3 grains), caffein ( $1\frac{1}{2}$  drams), ether (hourly,  $\frac{1}{2}$  to 1 dram), strychnine ( $\frac{1}{2}$  to  $\frac{1}{4}$  grain per dose). Also, to stimulate bowel peristalsis, use physostigmine ( $1\frac{1}{2}$  to 5 grains per dose, dissolved in 30 to 75 minims of water). But physostigmine, as also the other remedies, often fails to act. Internal stimulating remedies are also useful: wine, brandy, dilute alcohol; they are admirable analeptics, the same, too, are coffee and liquor ammonii. These remedies should be given either by a stomach tube or as clysters, and not as drenches. The commonest and most easily applied external stimulants are warm vinegar, oil of turpentine, spirits of camphor, mustard oil, croton oil, diluted spirits of ammonia, or inunction of ointment of tartar emetic (1 : 4), alternate warm and cold packings and fomentations, cold douches upon the head, hot flat-irons passed along the back upon an intervening blanket. Electricity is also said to have been applied with good effect. On the advantages of phlebotomy we possess experiences partially positive and partially negative.

2. To combat the suppressed activity of the bowels various purgatives are used. Here comes first, by reason of its mode of application and stimulating effect upon the muscles, physostigmine, and after it, pilocarpine, then tartar emetic ( $2\frac{1}{2}$  to 4 drams per dose), croton oil (20 to 25 drops per dose in mucous decoctions), aloes ( $1\frac{1}{2}$  to 2 oz. per dose), Glauber's salts (16 to 32 oz.). Peristaltic action is also assisted by repeated clysters and frequent exploration of the rectum, to remove all accumulated faeces.

3. To subdue any spasms or great excitement use morphia injections, clysters of chloral hydrate and bromide of potash, inhalations of chloroform, etc. The drinking of so-called anti-spasmodic decoctions (camomile, valerian, fennel, aniseed or caraway seed) is better left alone, owing to its danger. In

general, we recommend waiting as long as possible before slaughtering.

**NOTE.**—Since the introduction by Schmidt of the iodide of potassium treatment, all other methods have practically fallen into disuse. This method, which is based on the theory that milk fever is a toxæmia produced by a poison generated in the udder, and which is followed by about 90 per cent. or over of recoveries, consists in injection into the udder of four drams of iodide of potassium dissolved in thirty-five ounces of sterilised water (one-fourth into each quarter), after thoroughly washing and disinfecting the gland. Chinosol similarly used also yields good results, and lately inflation of the gland with filtered atmospheric air or oxygen has yielded even better results than either.

#### EXCESSIVE SEXUAL IMPULSE (*Nymphomania. Satyriasis*).

**Occurrence.**—Excessive sexual impulse in domestic animals is described under several names, such as wantonness, ruttishness, roaring sickness, seminal or maternal staggers and also—but very erroneously—as “hysteria.” It is most frequent in cattle, rarer in horses, sheep and dogs, still rarer in pigs and goats. This wantonness is seen most in cows, mares, male dogs and rams; other male animals, such as stallions, bulls and he-goats, are only occasionally affected. The reason why cows suffer so from excessive sexual desire lies in their lack of natural exercise, in their being accustomed to conceive at certain times, as well as in diseases of the genital organs caused by frequent parturition, in tuberculosis, etc. Nymphomania, as it is called in females, has been noticed to be more marked in some races of cows than in others. Of course, the term is only applied to abnormal excess of impulse, and no strict line can be drawn between a physiological and a pathological excess.

**Etiology.**—This does not constitute an independent disease, but is only a symptom arising from various causes.

1. In cows it is the older ones which are most disposed to it; also fattened kine, as well as those, four or five years old, that have been kept too fat.

2. In male animals, among whom it is called Satyriasis, the disease arises chiefly from too rich feeding, especially with rye, barley and leguminous foods, which are, therefore, termed “heating-foods.” Too little work and too much rest in the stable also conduce thereto.

3. Nymphomania is often produced in cows by pathological

changes in the generative organs, particularly by inflammation, cystoid degeneration, enlargement, dropsy, or by cancerous, sarcomatous and tuberculous new growths of the ovaries. The frequent occurrence of a morbidly increased sexual impulse as a sequel to tuberculosis led to this being at one time called "French-sickness." Nevertheless, it is not the tuberculosis, among these pathological changes of the ovaries, which is the chief cause, but the cystoid degeneration. Thus Schmidt found no tuberculosis in ten ruttish cows which he dissected. Tuberculosis, moreover, only causes wantonness when it attacks the genitals, and above all the ovaries. It is, furthermore, a mistake to imagine that the pathological changes in the ovaries must always be combined with nymphomania. The latter is, however, often found apart from any affection of the ovaries. Especially may nymphomania be caused by diseases of the uterus which prevent conception, by chronic vaginitis and endometritis, occlusion of the os uteri, new growths and displacements, atrophy and hypertrophy of the womb.

4. Among stallions, those which suffer most from excessive sexual desire are the monorchids and cryptorchids, in whom one or both testicles are retained in the inguinal canal or in the abdominal cavity.

5. Frequent sexual excitement, such as that produced in stallions by the vicinity of ruttish mares, or the constant inflaming of mares by stallions without gratification of their sexual desire (ending in the latter procuring self-gratification by onanism), leads in the end to respectively permanent satyriasis or nymphomania.

6. Immoderate sexuality is also observed during the outbreak of vesicles on the genitals of horses and cattle (pox), during maladie-de-coit in horses and also during rabies in dogs; Further, during certain affections of the spinal medulla, during gastric conditions or in plethora of the abdomen.

**Hysteria.**—The comparison formerly made between nymphomania of cattle and hysteria in woman is unjustifiable. Hysteria in man is a neurosis of the entire nervous system, including, especially, that of the brain, which often has nothing whatever to do with the generative organs. Even when it is caused by certain changes in the womb (catarrh, displacement, new growth, etc.), it is very rarely accompanied by symptoms of increased sexual desire. Hysteria manifests itself rather in melancholy illusions, hyperesthesia, spasms, anesthesia, paralysis, etc.

**Symptoms.—In Cattle.** Nymphomania begins in cows as follows:—Apparently healthy animals become lustful at

the usual period several times, but without conception, or their ruttishness breaks out at first every three weeks, later still more frequently, and finally grows to be constant. The cows show persistent excitement and disquiet, together with a continual desire to gratify their abnormal lustfulness. They try to approach other cows and to leap upon them and ride them, nay, they will even ride upon bulls. The strength of these impulses varies much according to the individual; when very stormy the signs are called "roaring sickness." This latter form seems more prevalent in some districts than in others. The affected cows manifest great restlessness, have a glaring look and very glittering eye, are easily startled, fond of leaping upon other cows, rub their horns on posts and troughs and dig with them into the earth, sometimes even in a kneeling posture. They also paw the ground like bulls, and utter at the same time a peculiar bull-like roar, which, beginning as a gentle lowing, rises to a loud, prolonged roaring noise, which is heard day and night, without any apparent cause, but is most frequent in the morning, or when any stranger enters the byre. Sometimes this behaviour proceeds to perfect savageness; the beasts seem frantic if any stranger approach, even snapping their chains, rushing furiously about and attacking men. Other signs which must be noted are: considerable falling off in the supply of milk, as well as coagulation of the milk when boiled; also a sinking of the body on both sides of the tail, as in highly pregnant animals. This is caused by relaxation of the sacrococcygeal ligaments, and makes the tail look as though inserted higher up. Then we note a gradually developing bull-like nature, whereby the muscles of the neck grow thicker; and finally, an increasing emaciation and feebleness, which may at last change to cachexia.

**B. IN MARES.**—Here also we notice at first an unusually extreme or oft-repeated ruttishness, which may not unfrequently become permanent, and in which the animal, though duly covered, does not conceive, or else regularly miscarries. She then appears very restless, of unsteady eye, neighs often, shows great desire to urinate and strains to do so, emitting only a small quantity of urine mixed with mucus in the form of a thick, whitish-yellow substance, which she squirts out with much force by rapidly turning over the labia pudendi and pressing forward the clitoris (so-called, flashing). She is, moreover, very ticklish and excitable. Every touch on the flanks, belly or hindquarters by the hand, halter, reins, traces,

or the legs and spurs of her rider causes squeaking, flashing, urination (in drops), lashing of the tail, counter-pressure, lowering of the hindquarters and even kicking and biting, so that she becomes dangerous to handle. The course of this disease is very variable and may be marked by temporary loss of appetite and gradual emaciation. It may at times develop into positive fits of spasms and maniacal symptoms. Thus Eletti noticed in a nympho maniacal mare pronounced signs of madness combined with tetanic spasms, grinding of teeth, difficulty of swallowing, contraction of the belly-muscles and palpitation, a condition not unlike that of hysteria in man.

Not unfrequently a considerable dulness of the sensorium shows itself later, a condition resembling sleepy staggers, and to which the name is usually given of maternal staggers. At the same time a periodical increase of these morbid symptoms is observed, corresponding with the occurrence of rutting. Although the signs just described will in some cases disappear for a time after the animal has been covered, yet they generally soon return again, and produce, by their prolonged excitement, a very prejudicial effect upon the mare's general health.

C. IN MALE ANIMALS.—Stimulated sexual instinct, the satyriasis of horses, manifests itself in frequent erection of the penis, which sometimes becomes persistent (priapism, *chorda venerea*), constant unrest and excitement, continual whinnying or roaring, great excitement at sight of a mare and repeated attempts to leap upon her. If this sexual desire be not gratified, the animal's condition will sometimes rise to perfect mania. It is said that bulls and stallions, when in this state, will pursue men, and especially women (Spinola). Later the animal becomes dull, refuses its food, wastes away and, according to several observers, develops paralytic signs in the spinal medulla, resulting in a staggering gait and a gradually increasing cachexia, which ends in death. In stallions sometimes, though not often, a condition resembling staggers appears, the so-called "seminal staggers," to which, according to Roll, stallions which have once been used for breeding purposes and then afterwards disused are specially pre-disposed. But the title "seminal staggers" is evidently applied in literature in various senses. Some understand thereby the signs of mania which appear in the course of satyriasis, and others a persistent condition of depression of the brain occurring during the same disease, which forensically should be compared to sleepy staggers. Lastly, it seems

that in some cases the viciousness developed during satyriasis has been erroneously termed seminal staggers.

Finally, satyriasis leads to attempts at self-gratification of the sexual impulse.

**Onanism.**—Quite apart from satyriasis, the habit of self-gratification, or voluntary loss of semen, is not at all uncommon among male domestic animals. This cannot be regarded as an outcome of abnormally stimulated sexual impulse, because stallions and bulls which are addicted to it remain perfectly indifferent to mares and cows. The practice is commonest in dogs and rams, but is not rare among stallions and bulls. The consequences vary much according to the degree of indulgence. In some cases the disturbances are scarcely noticeable; in others we observe gradually increasing symptoms of laziness, slight sweatings and fatigue, emaciation; dulness of the sensorium; weakness and signs of paralysis, arising from the spinal medulla; enfeebled power of procreation, amounting in time to complete impotence; cachexia, epilepsy and fits resembling apoplexy. In one such case of a stallion Spinola found the lumbar medulla softened for a distance of three inches. Treatment consists in punishment, hard work, short rations and admission to coition. If these fail, castration is the only remedy. Prangé cured the evil in a stallion by making it stand two hours a day for ten consecutive days in cold running water.

**Therapeutics.**—The most favourable cases of nymphomania and satyriasis are those which arise from too little work and too much food. The first thing to do then is to provide more work and less fodder, to separate the sexes and, if convenient, to gratify their sexual desires. Besides these, purging by way of the intestinal canal should be attempted, using Glauber salt for cattle, and aloes or tartar emetic for horses. One might even have recourse to blood-letting. Cold washings and cold baths should also be applied. The sexual impulse may also be diminished by means of drugs, the best of which are chloral hydrate (horses and cattle 5 to  $12\frac{1}{2}$  drams in their drink); bromide of potash, bromide of soda, sulphonal (3 oz. daily, for horses); also subcutaneous injection of morphia (3 to 7 grains for both horses and cattle).

If these remedies prove unavailing, there is nothing left but castration, unless perhaps we try the suggestion of Eloire, and introduce some foreign object, such as a leaden ball, into the uterus; or follow Schünhoff's plan of widening the uterine orifice. But the only certain cure for nymphomania and satyriasis is castration. In male animals and cows it can be performed without danger. But in mares the operation should

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only be undertaken when the animals have been proved to be otherwise useless, as there is risk of fatal peritonitis. But if carried out from the vagina, the operation should be free from danger, even in mares (Cadiot).

In cases of cystic degeneration of the ovaries in cattle and horses, some recommend that these cysts be crushed by the hand from the rectum (Zangger, Utz, Brüller and others). Utz says that of 100 cows he thus cured 90. But even after castration and crushing of the cysts, signs of restlessness and excitement may remain, especially if there be some simultaneous affection of the uterus, such as in tuberculosis. In the case of cattle it is best, under such instances, to slaughter them soon, even though in poor condition. For the same reason, it is not advisable to delay too long after the administration to cattle of internal remedies.

### *ABNORMALLY DIMINISHED SEXUAL IMPULSE.*

**Occurrence.**—This is a pathological appearance of great importance to all cattle breeders. It occurs mainly among females, and especially in cows and female yearlings, but also in the males. Sexual impulse may be abnormally diminished in various degrees, or may be entirely absent. Either it does not appear at the customary period, or only very rarely, or it is exceedingly feeble and lasts but a short time. If such lack of impulse be not merely transient, as is usually the case, but permanent, then it amounts in its results to sterility or unfruitfulness.

**Etiology.**—Among the causes of diminished sexual impulse the following are the most important :—

1. *Diseased Condition of the Sexual Organs*, viz., catarrh of the vagina and uterus (fluor albus), retention of the after-birth, hydrometra, pyometra, degeneration of the ovaries and testicles, faulty development or malformation of the same.

2. *Weakness of the Sexual Organs*, consequent upon using the male or female animal too early for breeding, too frequent copulation, misuse of stimulants, onanism.

3. *Torpidity of Sexual Life*, caused by a phlegmatic temperament, and great proneness to lay on fat. These two usually go together, and often arise from too rich and abundant diet combined with lack of exercise.

4. *General Condition of Weakness* after a prolonged course of

spare and poor diet, over-exertion in work, recent severe illness, affections of the spinal medulla; also acute feverish, as well as chronic, constitutional maladies.

5. *Psychic Disinclination* of the male to certain female animals, and especially to those of certain colours; *lasciness* in the male. There are also some fastidious stallions which much prefer covering maiden animals or gelded mares, and despise such as have foaled.

**Treatment.**—This, of course, must vary according to the originating cause. Fodder and keeping must be dietetically regulated. With enfeebled animals nitrogenous foods are necessary (oats, leguminous vegetables, rye, bread, raw eggs); and for fat ones, lessened fodder and abundant exercise. Stallions must either be freely ridden or used for heavy traction. Bulls also, if early accustomed to it, may be put to labour, or placed in a paddock by themselves. Finally, the regulation of sexual life must not be neglected. Avoid allowing too frequent or too early coition to the males, or too often bringing together phlegmatic females with male animals, or taking mares frequently to the stallion. Sexual impulse may also be stimulated by administering exciting remedies (aphrodisiacs). Among these cantharides stands first and, like all similar remedies, acts best when we have merely to deal with an abnormally enfeebled impulse, and should therefore only be given at that time. To large cows give daily, for several consecutive days, 30-75 grains offpowdered cantharides between two slices of bread or in wine, and to horses 15-30. Of the tincture of cantharides give cows 5 drams, horses 2½ and dogs 3-15 grains. For the same purpose all spicy, balsamic, resinous and alcoholic remedies, or such as contain ethereal oil, are useful, viz., pepper (2½-4 drams for cows), ginger, caraway seeds in wine, mustard-seed, juniper berries, aloes, savin tops, brandy, egg-flip, alcohol, etc. All these substances produce hyperæmia of the abdominal organs, turgescence of the ovaries and testicles, with consequent increase of sexual impulse. If, however, the causes lie in organic changes of the generative apparatus, then no treatment is of any avail.

#### IMPOTENCE AND STERILITY.

##### I.—IMPOTENCE IN MALE ANIMALS.

This impotence, or incapacity to propagate, may arise, on the one hand, from inability of the male to perform the act of coition

as such in correct physiological manner, in spite of the normal operation of the sexual glands; or, on the other hand, from incapacity to coitate fruitfully, although the accessory genital apparatus may perform its functions properly. For better understanding of the conditions we must differentiate, therefore, between "impotence of coition" and "impotence of procreation."

1. *Impotence of coition* may also be termed "relative" impotence viz., such as can be removed. It arises from disturbances which render preparation for the act of copulation or its performance more difficult, or even impossible. The cause lies, in such cases, in accidental external conditions. Impulse may be strong, and the semen fruitful; but weakness may be present in the genital apparatus (compare the paragraph on the causes of diminished sexual impulse), due to disturbed innervation consequent upon chronic affection of the spinal cord and brain. Probably the term "impotence" is best applied to such conditions of weakness. The animal cannot erect its penis nor introduce it into the vagina, or discharges before the erection is complete, or sometimes the erection is so brief that discharge occurs before entrance to the vagina. Other causes may be morbid conditions of the penis, e.g., kinking, phimosis, paraphimosis, new growths on it or abnormal size. Also motor disturbances, which prevent the male from leaping on the female, or render this impossible, such as painful affections of the joints, like spavin or ring-bone, or weakness in the hind-quarters, like the so-called broken back. Treatment in such cases must aim first at removing these primary causes, and should consist mainly in good food, electricity, in operative help and surgical remedies. Compare also the treatment for lack of sexual impulse.

2. *Impotence of Procreation* arises either from deficient production of semen, so-called aspermia, or from the absence therein of spermatozoa, so-called azoospermia. Here it may be that both testicles are lacking, or that they are atrophied in consequence of inflammatory and degenerative conditions, e.g., after carcinomatous destruction, aplasia, hyperplasia of the testicles, etc. There are, moreover, cases in which, in spite of apparent normal soundness of all the generative organs and especially of actual *ejaculatio seminis*, copulation remains unfruitful. Here it is the spermatozoa which are absent from the seed, a condition noticed in old stallions and sometimes from the very first in young ones. The fact may be established by micro-

scopical examination of the semen discharged. Cases also occur in which the spermatozoa are present, but in a motionless, that is, a lifeless condition. The cause of this is to be sought in chronic inflammatory or degenerative conditions of the seminal canals, or in congenital aplasia of the same. Treatment for impotence of procreation is quite useless.

## II.—STERILITY, OR UNFRUITFULNESS IN FEMALE ANIMALS.

This may consist in incapacity for the act of coition, or for fertilization or germ-formation; and may be either transient (curable) or permanent (incurable). The transitory form of sterility may arise from nymphomania, diminished sexual impulse, occlusion of the os uteri, or neck of the womb, by chronic inflammatory processes or scarred strictures. In cows and goats more especially, stenoses of the vagina, a misplacement or deviation in position or direction of the entrance of the vagina, displacement (kinking, twisting, inversion, prolapse) of the womb and new growths therein, may all produce this result; also chronic inflammatory processes of the mucous membrane of the vagina, uterus, tubuli, especially chronic endometritis (*fluor albus*) as a complication of parturition or of epizoötic abortion. A breaking-out of blisters or vesicles may also leave sterility behind. The treatment of all such conditions is purely surgical.

Incurable sterility can generally be traced to absence of the ovaries, or atrophied conditions of them. Among such must be named: aplasia, hyperplasia, atrophy of the ovaries, cystic, carcinomatous degeneration, occlusion of the ovarian cavity (in mares) by circumscribed peritonitis, which, by forming a deposit of connective tissue, hinders the eggs from leaving the ovary; further, defects in the uterus itself, aplasia and atrophy of the same, hermaphroditism in twin-births, etc. But after chronic endometritis incurable sterility may remain. Eloire produced artificial sterility in cows by introducing foreign objects into the womb, such as lead-balls and stones.

## MILK ABNORMALITIES.

**General Remarks.**—These abnormalities play an important part in the keeping of cows, and as the veterinary surgeon is often consulted about them, must be treated in detail. Our present information respecting milk abnormalities we owe largely to the researches of Fuchs, Haubner, Fürstenberg and

others. The causes of the various abnormalities are in general as follows :—1. Food which is abnormal either in quality or quantity. The fact that such conditions occur most frequently in cattle is accounted for by their somewhat unnatural mode of feeding. 2. Gastro-intestinal catarrh, cachectic and other morbid bodily conditions. 3. Diseases of the udder. 4. Infectious (bacterial), thermic, chemical and electrical influences acting from without. Apart from their veterinary aspect, milk abnormalities are important owing to their secondary action in provoking disease in man and other animals.

**Sub-division.**—Milk abnormalities may be variously classified according to different points of view.

1st. Those which occur as *anomalies of secretion* : agalactia, milk which is too watery or too fatty.

2nd. Abnormalities caused by *external influences*, especially the invasion of bacteria ; curdling milk, that which cannot be turned into butter, slimy, putrescent, soapy, blue, red and yellow milk.

3rd. *Milk abnormalities produced by foreign admixtures*, e.g., dirt, bitter and rancid substances, pigments, poisons, drugs, diseased matter and blood.

#### I. AGALACTIA. ABSENCE OF MILK. MILK STOPPAGE.

**Etiology.**—Arrest of milk is one of the earliest symptoms of internal disease, and especially of stomach or bowel complaints. It also arises from bad feeding, prolonged hunger, as well as from over-exertion in work. Certain anomalies of the udder must likewise be considered, viz., mastitis, especially the chronic catarrhal form (*Agalactia catarrhis contagiosa*), an imperfectly developed udder, fatty udder, congestion during the time of menstruation, etc. Psychic affections, such as pining for their young and for the accustomed stall, or fear occasioned by being milked by strangers, may all affect the secretion of milk. Agalactia is met with also in those bearing young for the first time, particularly mares. The bad habit contracted by some of sucking their own milk, or its being sucked by a neighbouring animal cannot strictly be reckoned as agalactia. In a few solitary cases the absence of milk cannot be traced to any cause. Atropine is said (?) to reduce the yield ; so also do the salts of iodine and external rubbings of rosemary oil and opodeldoc.

**Therapeutics.**—The so-called "milk driving" remedies (galactogogues) have always been recommended for the treatment of agalactia, together with a supply of good food as fluid in character as possible, such as scalded food, brewers' grains, etc. The favourite remedies are preparations of antimony (black and yellow antimony), sulphur, chlorate of potash, fennel, juniper berries, caraway seed, aniseed, dill, common salt, etc. These may either be given alone or in a great variety of combinations. As a so-called "milk-powder" we generally use the following mixture:—Black sulphide of antimony, 3 ounces; sulphur, 1½ ounces; fennel, caraway and juniper berries, of each 5 ounces; common salt, one pound. One tablespoonful of this mixture to be given to cow with each feed.

**Unusual Lactation.**—In opposition to agalactia, secretion of milk and swelling of mammary glands sometimes occurs in many animals in the absence of pregnancy. In single cases this has been observed to arise from the animal having fancied itself pregnant, or that it had given birth. Thus in bitches (pugs) we have often had occasion to notice lactation quite independent of either pregnancy or parturition. Among 70,000 dogs treated in the Berlin dog hospital, 65 cases of this unusual lactation were observed. The same occurs also in horses, cows, goats and sheep.

## II.—WATERY MILK.

**Etiology and Treatment.**—Watery milk is distinguished by an absence of fat and casein, and an excess of water. It is bluer in colour and of relatively higher specific gravity. It is caused by poor, watery food, as, for instance, an excessive or exclusive diet of mashes, turnips, turnip-tops, etc.; by disorders of stomach or bowels; by cachectic and hydramic conditions; and, at times, by some peculiar characteristic of the breed, quite apart from any of the foregoing factors. *Treatment* consists in change of food, supply of dry fodder and administration of stomachics, such as common salt, 6 ounces; caraway seeds, gentian and calumba, of each 1½ ounces. Of this mixture one tablespoonful should be given with each feed. But previous to this any morbid affections of the intestinal canal, blood, etc., which may be present, must be removed.

## III.—MILK THAT IS TOO FAT.

**Etiology and Treatment.**—This is found most frequently in sheep after too rich feeding, especially with leguminous food, and also during the rutting season. The result of drinking

this excessively fatty milk is to cause gastro-intestinal catarrh, diarrhoea, etc., in the lambs. The remedy lies in lighter provider for the ewes, avoidance of food which is too nitrogenous and in abundant exercise.

## IV.—CURDLING MILK.

**Etiology.**—Premature coagulation of milk—the so-called “curdling,” “sweet curdling” or “cheesing” of milk—is one of the commonest faults in cows’ milk, and occurs also frequently in that of goats. Its causes are very varied. On the one hand they arise from digestive disorders, sour food-stuffs, such as sour mash; from diseases of the udder, especially inflammation, from hyperæmia and swelling in advanced pregnancy; nymphomaniacal conditions; over-heating of the body by movement or work. On the other hand it is sometimes caused by excessive summer heat, or electrical tension of the atmosphere during storms; steamy stables, dirty utensils and straining cloths in the dairies. According to Fleischmann, feeding on corn infested with uridineæ will also produce “curdling milk.”

**Phenomena.**—These are that the milk curdles of itself within a few hours of milking, and especially that it coagulates in boiling. The evening milk shows greatest liability to this fault. The formation of acid is only very slight—hence the name “sweet curdling”—the amount of cream is small, so that it is difficult to churn the milk into butter. In mastitis the milk is already charged with flaky coagulae when drawn from the cow.

**Therapeutics.**—Treatment is regulated by the causes. Good ventilation and coolness in the byre, as well as perfect cleanliness of utensils and straining cloths, are imperatively necessary. Immediate cooling of the milk in refrigerators is much to be recommended, and also the addition of alkali, as much bicarbonate of soda as will rest on the point of a knife in each quart, or of salicylic acid (4 grains per quart). The internal administration of alkalis, especially soda, is also found useful; on the other hand, we cannot commend the use of acids internally, as some have done. As a remedy for curdling milk Harms administers fennel with sulphide of antimony (13 oz. of each; one-sixth part morning and evening). Diseases of the digestive canal, udder, etc., so far as these are the causes of curdling milk, demand special treatment.

**Causes of Coagulation of Milk.**—These lie in the action of a ferment discovered by Pasteur in 1857, the *lactic ferment*, which decomposes the milk-sugar into lactic acid, and thereby causes the acidulation and coagulation of the milk. Lister has called this organism the "bacterium lactis," and discovered that there are also many other micro-organisms which can produce these effects in milk. To these belong, according to recent researches: *osteomyelitis coccus*, *staphylococcus albus*, *citreus*, *cereus albus*, *cereus flavus*, *streptococcus pyogenes*, *bacillus pyogenes faecalis*, *erysipelas-streptococcus*, the bacterium, *micrococcus* and *sphaerococcus lactis acidi*, the bacterium *lactis aerogenes*, *micrococcus ovalis*, *streptococcus coli gracilis* and others.

#### V.—NON-BUTTERING MILK (FERMENTING, FROTHY MILK).

The causes of "non-buttering," "dumb," "frothy" or "fermenting" milk are to be sought in digestive disorders, in general disturbance or in bad and in-nutritious food, such as turnip-tops; also in udder-affections such as occur at the end of pregnancy; in the effects of great heat or cold; in the presence of other faults in the milk (curdling, rancid, putrid); and, lastly, in the presence of certain micro-organisms. Always pre-supposing, of course, that the faults do not lie in the method of making the butter. If the milk of a cow thus affected be mixed with that of a healthy animal, the latter milk is also infected. Milk which will not butter, or which ferments, is a cause of much trouble in dairies and cheese factories. According to Adametz, both bacteria and blastomycetes may produce this defect. The former generate gas at the cost of the casein, the latter at the expense of the milk-sugar. He isolated several bacteria, as well as a yeast-like fungus, the *saccharomyces lactis*, and proved their fermenting properties by experiment.

**Symptoms.**—The cream on milk which normally coagulates only during boiling is present only in a thin layer, and cannot be turned into butter at all, or only with great difficulty, as it coagulates, foams very much and refuses to collect. On long standing it shows small, yellow spots on the top, becomes greasy, pappy, and whatever butter has been secured is crumbly and very soon goes rancid.

**Treatment.**—In addition to the various milk remedies (fennel, antimony, caraway seed, etc.), the Haubner-Siedam-grotzky acids are very effective, viz., hydrochloric acid and acetic acid. We also recommend alum (40 grains thrice daily) and chalk (Harms) three times a day in doses of  $1\frac{1}{2}$  to 3 oz.

If the milk have a bitter taste, add  $\frac{1}{2}$  oz. chlorate of lime. Contamine commends a mixture of aniseed, antimony (5 drams each), vinegar and water (about 1 pint) for eight days running. All utensils and the byres must also be disinfected.

## VI.—PUTRESCENT MILK.

**Etiology.**—This abnormality of milk—which is very rare—is caused by infection of the milk by putrefactive bacteria. Infection is usually conveyed by dirty byres, stalls and utensils. But processes of decomposition in the intestinal canal, or feeding with rotten food (rancid bone-meal, for instance), may also produce it. The infecting micro-organisms are principally bacterium *termo* and *lineola*.

**Symptoms.**—On the top of the thin layer of milk small bubbles of gas, consisting of sulphuretted and carburetted hydrogen, show themselves after three or four days; these burst and leave indentations behind, in which the cream has vanished. The remaining cream gradually becomes of a dirty colour and mixed with drops of oil; its taste is bitter-sweet, foul, rancid (free fatty acids, especially butyric, valerianic, caproic, caprylic and capric acids). The milk refuses to turn into butter.

**Therapeutics.**—In this case prophylaxis consists in disinfection and cleansing of stalls, dairy and all utensils; in the internal administration of stomachic remedies and such as check fermentation, so far as processes of decomposition in the intestinal canal call for them. For this latter purpose the neutral salts, common salt, hyposulphite of soda and hyposulphite of lime should be tried.

## VII.—SLIMY AND STRINGY MILK.

**Etiology.**—In this condition the milk can be drawn out into long tough threads. It is the result of bacterial infection, but there is still some doubt as to the actual causal organism. Schmidt-Mülheim found, on placing such milk under the microscope, small, round, mobile bodies, which refracted the light and were about one micro-millimetre in diameter—partly in the form of micrococci and partly of beady chains. The material of fermentation is said to be the milk-sugar. Solutions of albumen or of casein, when mixed with the ferment, do not

produce fermentation, whereas a 1 per cent. solution of milk-sugar shows distinct mucous fermentation. The mucous matter, when extracted by alcohol, shows by its reactions the character of a vegetable mucus (similar to that of quinces); and the milk-sugar disappears at the same time completely. More recent investigations show that several micro-organisms possess the property of causing stringy milk. Hüppe and Loeffler have discovered and isolated, the one a micrococcus, the other a specific bacillus. Ratz differentiates between slimy and stringy milk. The slimy fermentation, according to him, is not the work of a particular micro-organism, but of several, among which he fully describes a diplococcus. Adametz cultivated a bacillus which rendered sterilised milk in the highest degree stringy.

This abnormality occurs most frequently in summer, and in dirty byres or dairies. Bad digestion, or feeding with rotting fodder infected with bacteria, may both be blamed. The consumption of pinguicula ("fat-herb") is said also by Müller to make milk stringy; and in Sweden this has been experimentally proved.

**Symptoms.**—Whereas, on the first day after milking, the milk is still thin, on the second it is thickly mucous, flows badly, draws out into threads, curdles badly and has but little cream, an insipid taste, is difficult to churn, and the butter obtained is moreover greasy and of bad flavour. It often presents a white, homogeneous mass, which is so tough that it will not run out when the bowl is reversed. On microscopical examination we find, in addition to the bacteria already named, that the casein has separated in the form of round discs (spherical crystals). Lately, slimy milk has been differentiated from stringy. The former produces a plentiful and thick layer of cream, which makes perfect butter. The stringy milk, apart from its different consistence, only forms a very little cream, or even none at all, and possesses a very pronounced power of infection, a small quantity thereof being able to infect large amounts of normal milk and to render it all stringy.

**Therapeutics.**—The most effectual preventive of this fault in milk is thorough disinfection of the milk chambers with sulphurous acid, hot steam, etc., and careful cleansing of all utensils. Schmidt-Mülheim says that heating the milk to 150° F. destroys the micro-organisms.

In cases of digestive disorder administer internally the

proper stomachics and anti-catarrhal remedies. Feeding with hemp-leaves is, according to Haubner, much to be commended.

#### VIII.—SOAPY MILK.

**Causes and Treatment.**—By "soapy milk," is understood such as has a soapy taste, which never coagulates, no matter how long it stands, but precipitates a slimy sediment and gives a very frothy cream, which is extremely hard to churn into butter. The causes of this milk anomaly are, according to Weigmann and Zirn, traceable to the presence of a special bacillus, the so-called *bacillus saponacei*. These bacilli are small motile rods, with rounded ends and measuring 1.6 micro-millimètres in length by  $\frac{1}{2}$  mm. broad. They liquefy gelatine and produce on it round, white colonies with a central yellow spot. When placed in milk they produce in the course of a few days a slimy condition and the characteristic soapy taste. They are supposed to be conveyed in straw and hay, both these forms of fodder being found to contain them. *Treatment* consists in removing the straw and fodder and in washing the udder.

#### IX.—BLUE MILK.

**Occurrence.**—Blue milk is observed most frequently in spring and the height of summer, particularly during moist, warm weather and in dark dairies. In the latter it may occur year in year out, even for ten years (Steinhoff). On the other hand, this condition generally disappears in the colder seasons, or after the air has been cleared by heavy rain. At first only one cow gives blue milk, but soon all the cows in a byre are infected. Very often sick cows show a pre-disposition to the production of blue milk.

**Etiology.**—Blue milk is caused by a special chromogenic bacterium, the *bacterium syncyanum* (Schröter). Fuchs described it in 1841 as *vibria cyanogenus*, and Ehrenberg gave it the name of *vibrio syncyanus*. According to Hueppe's more recent investigations, it occurs in the form of small rods, from two to four micro-millimètres long by  $\frac{1}{2}$  thick, which are motile, have rounded ends, are colourless and multiply by division and formation of spores. In sterilised milk (in which the bacteria acidilactici are killed) the bacilli only produce a slaty grey, or, at most, pale blue colour, owing to the absence of these bacteria.

and of the sour reaction which especially causes the blueness; whereas on peptone gelatine broth, it produces a green, which gradually changes to brown, and by oxydising means may be converted into blue. On sterilised potato the colour is greyish-blue. This very motile fungus decomposes albumen into a colouring matter very much like aniline blue. Its transmission takes place through the air and probably also by flies. Whether the milk can be infected by these bacilli while still in the udder is still uncertain. Milk which is rich in albumen, the result of feeding with fodder containing much protein, is predisposed to turn blue, as also is very alkaline milk which coagulates slowly or incompletely, in which respect it differs from acid milk. Haubner maintains that a sudden change of food or place of pasturage conduces to blue milk, especially if the latter have been manured with gypsum, and the appearance vanishes on removal to lighter pastures. Hueppe also believes that certain methods of feeding, diseases of the udder, etc., all have a secondary influence upon the blueness of milk by delaying acidulation.

**Symptoms.**—These, according to Fuchs, are as follows:— One or two days after milking, spots appear, simultaneously with acidulation and coagulation, upon the cream, at first light or greyish-blue, then indigo to sky-blue. They are small and irregular, the size of a pin-head, and gradually enlarging, penetrate later the layer of cream and finally also the deeper mass of milk, even turning it all blue. The consistence of the milk also changes, owing to the modifications wrought in its albuminous and fatty constituents. The casein loses in firmness and becomes more fluid, and the butter turns tallowy, rancid and discoloured. The blue discolouration, which appears in the form of spots or stripes, is often surrounded by a greenish, shiny border. After several days this blue milk decomposes, and putrefactive as well as pathogenic bacteria are developed, and the whole assumes a dirty grey colour. The consumption of blue milk is injurious for animals, as well as for men, though diverse opinions are held on this point.

**Treatment.**—Milk can be prevented from turning blue by ventilation, cleansing and disinfection of the byre, dairy and all utensils. The best disinfectant is freshly generated sulphurous acid. According to Haubner, an addition of sour buttermilk may replace any other prophylactic. In some cases

a change of food is advisable. Zürn suggests also washing the udder with antiseptics to destroy the bacilli.

## X.—RED MILK.

**Etiology and Symptoms.**—Like blue milk, this also is caused by the presence of bacteria. Several kinds of bacteria possess the property of turning milk red. So far the following are known :—1. *Bacillus prodigiosus*, discovered in 1838 by Ehrenberg, the cause of so-called "bleeding bread" and "the bleeding host." The bacilli occur in the form of very short, motile rods, which are often mistaken for cocci. When cultivated on potato at 68° Fahr., they quickly produce a blood-red covering, while, on inoculating sterilised milk on its creamy surface, red dots appear, which run together and finally dye the whole area red. No discolouration of the entire milk occurs, because the development of the bacilli depends upon the presence of oxygen and is therefore confined to the upper surface. Both at lower (43–50° Fahr.) temperatures and at higher, the cultivation is white. When dissolved in alcohol and ether, the pigment shows in the spectrum three characteristic lines of absorption (one behind D and one each before E and F). The red solution is bleached by alkalis. Although very widely disseminated, the *bacillus prodigiosus* only occasionally causes red milk.

2. *Bacterium lactis erythrogenes*, discovered by Hueppe in 1887, which, contrary to the preceding, stains the whole of the milk red. It occurs in the form of short rods with rounded ends 1–1.4  $\mu$  long by 0.3–0.5  $\mu$  thick, which produce a rennet-like coagulation of the milk accompanied by an alkaline reaction. The colour is formed only in the dark, and in the course of two to three weeks the entire milk becomes blood-red. The pigment is insoluble in alcohol and ether, and possesses two characteristic lines of absorption between D and E and a third in the blue part of the spectrum.

3. A kind of *Sarcina* also turns milk red (brownish red), as shown by Adametz. Probably there are other bacteria with the same property. Red milk should be prophylactically treated with disinfectants in the same way as the blue.

**Yellow Milk.**—This is also nearly related to blue milk. The colourless, mobile organism, *bacterium synanthum* (Schröter) produces a pigment like aniline yellow, ranging from yolk-yellow to lemon, which sometimes appears along with green and blue, and is more frequently seen

in boiled milk than in unboiled. Lactic acid prevents the development of the ferment. According to Schröter, after 48 hours a yellow, spotted discolouration appears in the boiled and already coagulated milk, with simultaneous alkalinity of the upper surface, and increase of the whey-fluid and of softness, along with disintegration of the milky mass into small coagulae. After six days the milk is changed to a lemon-yellow, watery fluid, very poor in caseous flakes. There seem also to be other bacteria which produce a yellow colour in milk.

#### XI.—FOREIGN MATTER IN MILK.

Of foreign substances in milk the most important are : dirt, abnormal flavouring matter, scents, pigments, drugs and poisons, also morbid substances such as blood or pus. The various bacteria already named, and which are the cause of many milk abnormalities, must also be included in the list.

1. *Dirt.* This comes from the cow's udder, which is often smeared with dung, and consists therefore almost entirely of cow-dung. If the cow be diseased (diarrhoea, enteritis) infection may be conveyed to those drinking the milk. Uhl found, for instance, from  $\frac{1}{2}$ -2 grains of cow-dung per pint of milk taken in the market at Giessen. Prophylaxis consists in cleansing the udder prior to milking, and in supplying dry straw. No cows' milk can be considered hygienically clean but such as, after standing for two hours in a vessel with transparent bottom, shows no sediment (Renk).

2. *Abnormal flavouring matter.* Such matter arises from food-stuffs consumed when in process of decomposition. We may mention the following : rancid oil-cake, bad or frozen potatoes and turnips, sour brewers' grains, or large quantities of turnip-leaves or turnip-tops. According to Schumacher, these contain an aetherial oil. Also ensilage and fodder that has long lain in a heap, leaves of trees, chestnuts, dandelion, wormwood, tansy, garlic, artichokes, thistles, the refuse of rape and linseed, an excess of oat or pea straw, olive oil, rape oil, linseed oil, fish refuse, etc. Dirty vessels and utensils may also cause this defect. In all these cases the milk obtains an unpleasantly rancid, bitter, sweetish, salt or harsh flavour. Jensen states, moreover, that the *bacillus foetidus lactis* produces an offensive rancid smell in the milk, with an oily flavour of turnips. Less disagreeable are the aetherial oils of the aromatic drugs : aniseed, caraway, fennel, etc. The remedy lies essentially in change of food and in keeping all vessels, etc., clean. Recent investigation shows that bitterness is produced in milk by

bacteria. Krüger blames for this an organism called *proteus vulgaris*; Hueppe says it arises from formation of casein-peptone by the *bacillus butyricus* and the *potato-bacillus*. Weigmann has proved a *bacillus* and Conn a *micrococcus* to cause bitter milk, so that evidently several bacteria possess this property.

3. *Strange odorous matter* gets into milk after feeding on onions and garlic, or after using highly-scented drugs, such as *asafotida*, *camphor* or *turpentine*. Odour is also largely absorbed from the entire environment, e.g., after disinfecting a byre with carbolic acid. In one case recorded, milk became so strongly impregnated after standing in a place thus disinfected, that a dog drinking it showed signs of carbolic poisoning (Zorn).

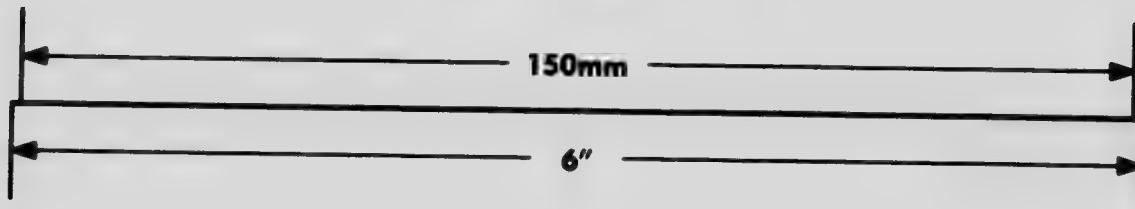
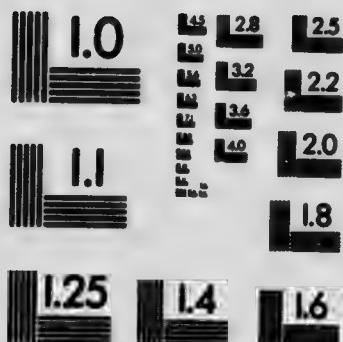
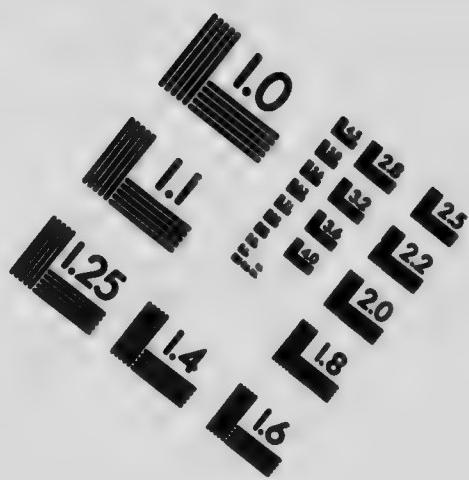
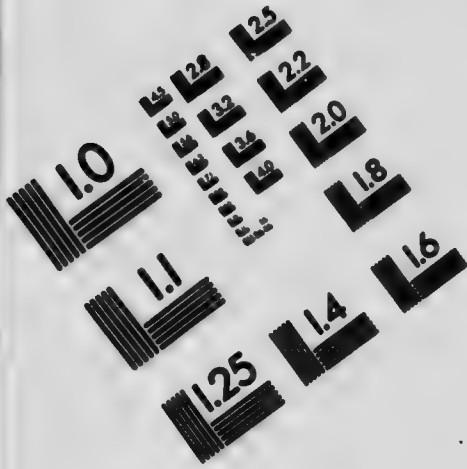
4. Of *Pigments*, yellow, yellowish-red and blue are met with. The yellow and red come partly from certain plants, e.g., carrots, rhubarb and crocus; and in part they are explained by admixture of biliary pigments (*icterus*), blood and pus in the milk. Moreover, in certain inflammations of the udder a yolk-yellow colostrum occurs in the milk. The blue pigments—apart from so-called "blue-milk"—have their origin in certain plants, such as *alkanet*, *horsetails*, *dog's mercury*, *buckwheat*, *flowering rush* and plants containing *indigo*.

5. Among *Drugs* the following pass into the milk, or at least their chief constituents, and produce corresponding effects upon those drinking it: *camphor*, *ether*, *oil of turpentine*, *asafotida*, *chloroform*, *tartar emetic*, *aloes*, *arsenic*, *hellebore*, *belladonna*, *henbane*, *stramonium*, *colchicum*, *corn-cockle*, *castor-oil cake*, *hemlock*, *morphia*, *strychnine*, *veratrine*, *euphorbium*, *senna leaves*, *salicylic acid*, *carbolic acid*; the various salts (*Glauber*, common salt, soda, potash, sulphate of magnesia, borax, etc.); the compounds of *bismuth*; salts of *lead*, of *copper*, *zinc* and *iron*; the compounds of *mercury*; *iodine*.

6. The *products of disease* are not unfrequently transmitted through milk to men and animals. The most important instance of this is the *transmission of tuberculosis in milk*. This has certainly been demonstrated in man. Moreover, experiments in feeding with the milk of cows suffering from bovine tuberculosis (see Vol. I., p. 185) have proved its power of infection to be very great. Friis found that 14 per cent. of the milk of commerce contains the bacilli of tuberculosis, and that mixed milk does not protect from infection. According to Lehmann, the milk of tuberculous cows is also poorer in fat and albumen.



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That of cows suffering from *pulmonary disease* is thick like colostrum, of a peculiar flavour and soon turns putrid. Haukold saw men vomit after drinking it. In *foot and mouth disease*, which is often communicated to men and animals through milk, Herberger found the milk imperfectly coagulated, viscid, slimy, resembling colostrum, bad in taste and smell, poorer in sugar and casein and sometimes containing ammonium carbonate. In *rinderpest* the fat is much diminished in quantity, and the sugary contents almost nil. Milk is also said to be capable of transmitting typhus, scarlet fever, cholera and diphtheria to man.

7. *Blood*, that is to say, *haemoglobin* in milk, can be traced to several causes. Not unfrequently blood may be noticed in the milk immediately after calving, and lasts for about fourteen days. In other cases it may be traced to mastitis, injury inflicted on the udder by a blow, rough milking or sucking, congestion of the udder during calving, irritation by substances containing turpentine, *haemoglobinæmia*, a rapid change to rich, concentrated and nitrogenous foods, especially clover, etc. The phenomena consist in uniform (*haematuria*) or streaky discolouration (red) of the milk (mastitis), formation of a sediment of red coagulae, etc. The treatment is partly local, partly general. For further details on "bloody milk" see the text-books on obstetrics; and for details on milk-changes produced by mastitis, consult those on surgery.

**Bacteria.**—Certain bacteria which appear in milk are scarcely to be classed as foreign substances. Besides the acid-forming bacteria named in connection with curdling milk, the following, according to Marpmann, also occur in milk: a *bacterium lactis acidi*, a *sphaerococcus lactis acidi*, a *micrococcus lactis acidi* and a *bacterium limbatum lactis acidi*. Of organisms which do not produce acidity the commonest, according to Loeffler, is the potato-bacillus (*bacillus mesentericus vulgaris*), then the gum-bacillus (*bacillus liodermos*), a bacillus of butyric acid, a white bacillus and finally the mould-fungus (*oidium lactis*, *penicillium glaucum*, and some kinds of *aspergillus*). The investigations of Cnops, Freudenreich and others have shown that in the cleanest of dairies and with the very best milk, samples taken from the large cans a few minutes after milking contain 100,000 germs per cubic centimetre. In six hours this enormous figure rose to six millions of germs per cubic centimetre! By passing the milk through a centrifugal machine it may be freed from some portion of its bacteria. Thus Wiss has shown that the scum deposited on the sides of the machine is seven times richer in bacteria than the milk itself which has been so treated. This process is therefore of importance for the purifying of milk from infectious matter, such as tubercle-bacilli (Bang, Scheuerlen).

**Milk Grit.**—A certain milk abnormality is so named in Switzerland, in which the otherwise quite normal milk shows upon the hand when milking fine, white, soft coagula or flakes. These arise from dried milk in the orifice of the teat, and are consequently found in the milk first drawn (Guillebeau and Hess).

**Infectious Agglactia.**—This infectious drying-up of the milk in sheep and goats, described by Oreste, Brusasco, Schlossleitner and others, and which is, moreover, complicated with lameness, blindness, etc., belongs evidently either to the sphere of epizootics, or of poisoning.

## CHAPTER IX.

### DISEASES OF THE HEART AND LARGER BLOOD VESSELS.

#### INFLAMMATION OF THE PERICARDIUM. PERICARDITIS.

**General Observations.**—This condition is very common in cattle, but rare in other domestic animals (horse or dog). Pericarditis in cattle is distinguished from the same condition in other animals by being usually traumatic in origin, and frequently complicated with inflammation of the muscles of the heart or myocarditis, as a consequence of which the whole aspect of the disease is changed. A similar traumatic pericarditis occurs also, though only rarely, in goats, but in horses and other domestic animals the condition is usually idiopathic. Consequently, in discussing inflammation of the pericardium, a clear line of division must be drawn between the traumatic pericarditis of cattle and goats and the idiopathic pericarditis of the other domestic animals, and this both on clinical, anatomical and therapeutic grounds.

#### I.—PERICARDITIS OF CATTLE. TRAUMATIC PERICARDITIS AND CARDITIS:

**Etiology.**—That this is a very common disease among cattle and by far their most frequent heart-affection, is proved by the rich and yearly increasing literature upon the subject. Nay, the question may even be asked: Is there any non-traumatic pericarditis among cattle? *A priori*, we must answer, yes, to this question, for the pericardium of an ox may suffer inflammation from cold, or in the course of acute muscular or articular rheumatism, in infectious diseases such as septicæmia, or in connection with parturition or pleuritis, just the same as that of a horse. All these several processes have been indicated, in

addition to wounds or bruises, as causes of pericarditis. And yet one often gains the impression that most of the cases described in literature as "rheumatic" pericarditis were really of traumatic nature. It seems to have been a general custom among observers to reckon all cases of inflammation of the pericardium, in which dissection revealed the presence of no foreign substance in the heart, as simply of rheumatic origin, forgetting that sharp-edged objects may work their way out of the heart into the second stomach. In any case, the greatest care is needful in diagnosing rheumatic pericarditis in cattle. Somewhat more frequent—though rare as compared with the traumatic form—may be the inflammation set up in the pericardium in the course of septic metritis, but here it is generally only one symptom among many other organic disturbances. Respecting the pericarditis which occurs during bovine tuberculosis, see the chapter on tuberculosis. Concerning the entrance and nature of the sharp-edged foreign bodies and their penetration from the second stomach, we refer our readers to the etiology of traumatic inflammation of the stomach and diaphragm (Vol. II., p. 95). We would here merely mention the anatomical fact that the second stomach is only a short distance from the pericardium and separated from it only by the diaphragm. This fact explains the frequent injury of the heart and its enclosing membrane by sharp foreign bodies. Pericarditis caused by wounds inflicted through the ribs, e.g., by goring or fractured ribs, is extremely rare. In one case on record the broken blade of a fleam was found in the heart (Kolb).

**Post-mortem Lesions.**—The changes in the pericardium and heart in traumatic pericarditis and carditis are usually very far advanced, so that the early stages and development cannot be followed. We generally find in the pericardium an exudation of fluid or solid nature, adhesion of the heart to the pericardium, a thickening and expansion of the latter and changes in the muscles of the heart, as well as secondary changes in the lungs, diaphragm, etc.

This pericardial exudation is very varied in its composition. Sometimes it is purely serous, then fibrinous, purulent, haemorrhagic or ichorous, and its colour is accordingly sometimes pale and clear, then milky or whey-like, sometimes whitish, flaky, or greyish-yellow, yellowish-green, reddish, brown, dirty red, etc. Its formative elements also vary much beneath the microscope, red and white blood-corpuscles, drops of fat and bacteria being

frequently present. The fluid masses are often mingled with putrid gases, which have either been generated in the pericardial sac itself or have penetrated thither from the second stomach, and are mostly more or less foul in smell, though at times quite odourless. The quantity of this exuded matter may be quite considerable, enough to fill a stable bucket (2—3 gallons). Great pressure is exerted thereby upon both the heart and its adjacent organ, the lungs, it compresses the auricles and larger veins and greatly distends the pericardium. If much prolonged, a chronic dropsy of the pericardium (hydro-pericardium) may develop. More rarely we find blood in the pericardium, which has reached it after perforation or rupture of the cardiac walls, and which may amount to some quarts.

The solid exudate consists of fibrinous, gelatinous coagulæ, which become organised later into connective tissue. They occur in the form of deposits of filamentous, string-like proliferations between the heart and pericardium. The inflammatory coatings which overlay the heart are particularly characteristic. These present thick deposits of yellow colour (varying from that of the yolk of an egg to that of an omelette), possessing a villous, nodular and even warty surface, and make the heart look exceedingly large, and its surface as though gnawed and hacked (shaggy heart, *cor villosum*). The epicardium itself is thickened and changed into a leathery membrane, which is sometimes incrusted with salts of lime, and envelops the heart as in a rigid cover. The coagulæ lying on the heart sometimes become slimy and undergo fatty degeneration in their lower layers. The solid exudation adhering to the heart and pericardium is of stringy, ribbon-like or tendinous character, and partly inter-penetrated by newly-formed vessels. Sometimes there is a complete union of the heart and pericardium. Occasionally after retrogression of the exudation in the pericardium, so-called macula lactea are left behind, viz., indurated, cicatricial thickenings.

The heart itself shows serous infiltration, with circumscribed fatty degeneration and softening of its muscular fibres; also inter-muscular abscesses, as well as cavities lined with abscess-membrane, which represent the space formerly occupied by the foreign body or else are mere losses of substance. They are then ulcerous, dark red in colour and surrounded by a yellowish border (wounds in the heart). The foreign object is sometimes found in the lumen of a ventricle, at others it lodges in the cardiac wall, or lies in the pericardium. In one case Camoin

saw a canal which ran from the left ventricle to the second stomach, through which the blood poured directly from the one into the other. In prolonged cases the heart becomes hypertrophied and dilated, or else, in consequence of pressure by the pericardial exudation, is atrophied, relaxed and small. The pericardium may, in course of time, become as thick as a man's thumb; it is also frequently much distended, so as to contain three gallons and more of liquid. We also observe pleuritis, adhesion and circumscribed traumatic inflammation of the lungs, adhesion of the pericardium to the diaphragm, and also—in traumatic inflammation of the stomach and dia-phragm—we find certain local anatomical changes.

**Symptoms.**—The aspect of traumatic pericarditis forms a continuation of the phenomena described in traumatic inflammation of the stomach and diaphragm. To sum these up briefly, they consist in chronic gastric disturbance without any assignable cause and with extraordinarily inconstant and changeable course, complicated with pain in breathing, in straining or in the passage of faeces, or on palpation of the diaphragm; also intestinal haemorrhage and great emaciation. The symptoms defy all attempts at relief by drugs, no matter how long continued. Along with these evidences of chronic gastric catarrh, and not rarely after an apparent diminution in their force, striking and quite characteristic heart and lung symptoms appear, accompanied by general disturbances. These usually come on rapidly, and sometimes after a previous birth or exhausting march (driving to market, etc.).

**I. Phenomena of the Heart.**—At the outbreak of the inflammatory affection the heart-beat is stronger, palpitating, jerky and even tumultuous, so as to be audible three or four yards away; sometimes a distinct back-stroke can be heard. Later, as the exudation accumulates in the pericardium, it often ceases entirely, or is very much weakened, but more diffused, feeling like the undulations of waves. Percussion of the heart, which may be very painful, as is that of the entire region of the heart, demonstrates an increase of cardiac dulness proportionate to the growth of the pericardiac exudations. Moreover, we not unfrequently notice, as one of the first symptoms, a tympanitic tone spread over the region of the heart, which is of great diagnostic importance, being due in most cases to separation of the heart from the pericardium by the interposition of exuded fluid,

and to accumulation of gas in the pericardial sac. This is partly generated by decomposition of the exudations therein, but comes also in part from the second stomach. Here and there the tympanitic tone extends over the entire left wall of the chest (pneumo-thorax, pleuritis, compression and atelectasis of the lungs). Palpation of the heart by the hand reveals in dry pericarditis a distinct friction, which keeps time with the action of the heart; where exudation is present, wave-like movements can sometimes be felt.

Auscultation of the heart is further of the greatest possible pathological importance. The ear must be placed near the fifth rib, or behind the left shoulder-blade or humerus, over the triceps. It is also advisable sometimes to try the right side as well. We then hear the true heart-sounds perfectly clear, although perhaps less loud and distinct owing to other sounds. Secondary sounds after the first or second heart-sound are exceptional, and indicate valvular disease and endocarditis. On the other hand, we hear pretty regularly in all cases the pericardiac sounds which are characteristic of pericarditis. These are of very different nature, according to the composition and amount of the exudation. At first, and so long as there is not much fluid in the pericardium, and the exuded matter is of comparatively dry and fibrinous composition, we perceive a decided rubbing sound, which is caused by contact of the two rough inner layers of the pericardium. Along with this friction sound we also gain the impression of creaking, grinding, grating, humming and stroking. In general, this rubbing noise is not heard very often, as it only occurs in plastic-exudative pericarditis, and then only at first. More frequent, and corresponding to the more usual fluid character of the exudation, are the sounds of liquid in the pericardium. When this is moderately full, they are splashing noises, which can be heard some yards away; or again, they are like clapping, gurgling and clucking, a kind of frothy rattle; or at other times the sounds have a metallic ring, like that of a falling drop, or are quite dull and fluctuating. This latter is when the pericardium is becoming over-filled. Whereas the rubbing noises are strictly connected with the pulsations of the heart, the other sounds may either precede or follow them, and at times cease altogether for a while. They all alike cease on re-absorption of the exudation, and in general synchronise with the heart-beats.

**2. Phenomena of the Lungs.**—The compression of the

auricles by the pericardial exudations, combined with the diminished efficiency (insufficiency) of the heart, soon produces a congestion of blood in the lungs, a passive hyperæmia of that organ, which shows itself in quickness of breathing, especially after movement, and in slight fits of coughing, and may sometimes lead to violent action of the breathing apparatus of dyspnœic nature. The symptoms of a lung affection then become prominent, and may be mistaken for an independent pulmonary disease, such as inflammation, phthisis, or even tuberculosis. As complications of the pericarditis with traumatic pleuritis and pneumonia, caused by the same foreign body, may also arise, the diagnosis is at first very liable to be erroneous. But a careful examination of the heart will soon correct any such error.

**3. Phenomena of the Jugular Vein.**—With the appearance of the pericardial sounds, that is, about 4-6 days later, and often not for 8-14 days, the jugular veins also become very highly charged, owing to the pressure exerted by the exudation upon the right heart, whereby the blood in them becomes engorged, and they show—though by no means always—a so-called "venous pulse," i.e., distinct movement synchronising with the action of the heart. At the same time doughy, cold swellings develop in their vicinity, which, when the head is raised, appear only on the mouth, but when it is sunk or stretched out, are found around the larynx as far as the lower jaw and cheeks, and at times extend between the forelegs to the breast and belly. This congestive œdema occurs also in other affections of the heart, but, when combined with the other phenomena of the heart, offers a very characteristic symptom of traumatic peritonitis, although one which is not found in every single case.

**4. General Phenomena.**—The irregularity of the heart's action is also transmitted to the pulse. This is at first very rapid (up to 120 a minute), then very weak, often thready, and at times irregular. The speed varies meanwhile with the intensity of the disease. Temperature is also very changeable, a circumstance in some respects important for the diagnosis of traumatic pericarditis; it may rise to  $106^{\circ}$ - $107^{\circ}$  Fahr. The temperature of the exterior also varies often, the extremities especially being first cold and then warm. The animals also show shiverings and trembling. In advanced cases the general condition is seriously affected. The creatures are very list-

less, appear anxious, have a pitiful look, and groan when they lie down, move, breathe, etc. The body is stiffly held, the neck held out straight, and the elbows stand out from the sides of the breast. All movement, especially sideways, is avoided, the animals lie down a good deal, keep getting up and lying down again, or cannot be brought to their feet at all. The visible mucous membranes are at first more highly reddened; later they become pale, cyanotic or yellow. Emaciation steadily progresses; hydræmic symptoms set in, and now and then intestinal haemorrhage appears, the animals perishing of exhaustion or asphyxia, after having, in some cases, also shown pleuritis in addition to pericarditis. Sometimes, towards the end of the disease, the complete aspect of pyæmia is shown, with metastasis in the inner organs (brain, spinal medulla, kidneys, liver, muscles, joints and tendon-sheaths).

The *duration* of the ailment is generally a long one, lasting for months, and the course very slow, interrupted by periodical exacerbations. More rarely death follows quickly in a few days, in consequence of perforation of the heart and haemorrhage into the pericardium. In a few cases recovery is seen, as, for instance, when the foreign body slips back into the second stomach. The time which elapses between the inflammation of the stomach and diaphragm and the pericarditis may be weeks or months. Much depends upon the nature of the foreign object, how the animal is used, its feeding, the number of times it bears young (advance of the foreign object by labour pains) and accidental circumstances. Much thickening and adhesion of the pericardium point to a duration of the condition extending over months.

**Differential Diagnosis.**—The diagnosis of traumatic pericarditis is not very difficult, if the examination be thorough. The previous chronic digestive disturbances, abnormal activity of the heart, the appearance of characteristic pericardial heart-sounds, venous engorgement, œdematosus swellings, combined with the chronic and finally fatal course (in most cases) offer safe grounds for a sound judgment. At the commencement of the condition the coincidence of three factors is of great importance for diagnosis:—1. Incurable chronic gastric catarrh; 2. tympanitic tones in the region of the heart; 3. disturbed breathing (shortness of breath, cough) in the absence of any demonstrable change in the lungs themselves.

**Therapeutics.**—No time should be wasted in treating traumatic pericarditis and carditis, but if its presence be positively established, let the animals be promptly slaughtered. On this point all opinions concur. Only at first, and so long as there is a slight hope of improvement and no immediate danger of death, or if the diagnosis be doubtful, one may attempt to regulate the action of the heart by administering digitalis (30-75 grains per diem), caffein (1½ drams), or tincture of strophanthus (2½-6 drams); or to stimulate peristalsis (tartar emetic, saline remedies), so as to remove if possible the foreign body. Such efforts are, however, usually without result. Better perhaps would be a surgical operation, consisting either in puncture of the distended pericardium, or removal of the offending object therefrom, where it may possibly be palpable or visible (Bastin). Whether the operation carried out and recommended by Meyer be of any use, if inflammation of the pericardium has already supervened, is doubtful, viz., the opening of the first stomach by laparotomy and manual removal of the foreign body.

## II.—PERICARDITIS IN HORSES AND THE OTHER DOMESTIC ANIMALS.

**Etiology.**—In contrast to the pericarditis of cattle, that of horses, dogs, sheep, etc., is in the vast majority of cases idiopathic, that is, produced by inner causes and not by wounds. Frequently these are of rheumatic nature and traceable to chill, to which horses are peculiarly liable. Schindelka has often observed pericarditis in horses after a long and uninterrupted railway journey. Or else it is developed in the course of general infectious disease, such as acute muscular and articular rheumatism, or septicæmia, phthisis or pneumonia. It may also arise from the spread of inflammation from an adjacent organ, especially from endocarditis, pleuritis, myocarditis and pneumonia, or after an empyema caused by perforation of a lung-abscess, in which case it would first appear as an external pericarditis. Finally, it is in horses sometimes traumatic in nature. Thus, Henniges saw a sewing-needle, which had penetrated from the pharynx of a horse into the left auricle; Lewin, in the pericardium of another, found a darning-needle, which had probably made its way from the duodenum through the diaphragm. Broken ribs and small shot may cause pericarditis in dogs.

**Post-mortem Lesions.**—The first pathological changes in pericarditis are injection, ecchymosis, opacity and swelling of the inner serous layer (pericarditis interna), along with a deposit of soft, thin and gelatinous exudate, which forms an easily detachable film. According to the origin we may find in the pericardium a serous, fibrinous (chills), haemorrhagic, purulent or ichorous exudation (infection, sepsis), in larger or smaller quantities (up to six gallons in a horse), from which, if the malady be prolonged, hydropericardium may develop, particularly in dogs and sheep. Later on there appear leathery, nodulated coatings upon the pericardium (*cor villosum*); adhesion of the pericardium to the heart; formation of encapsulated cavities between the heart and pericardium, and hypertrophy or even atrophy of the heart. Prakke found the heart in one horse no larger than a child's fist, and its walls in some places no thicker than a playing-card. In other respects, the appearances are described as mainly those described in pericarditis of cattle.

**Symptoms.**—These show themselves in horses and the other domestic animals at first in palpitating, tumultuous beating of the heart, which later diminishes as the exudate increases; in extension of the cardiac dulness upwards and backwards; in pericardiac, rubbin; sounds (friction fremitus); very frequent, small, irregular and unequal pulse; strained and even dyspnoëc breathing; jugular pulse and rise of temperature ( $102^{\circ}$  to  $105^{\circ}$  F.). At times we can perceive nothing characteristic beyond the high fever, imperceptible heart-beat and difficult breathing. *The course* is sometimes acute, when death may suddenly ensue in consequence of rupture of the heart; at other times it is chronic. In dogs we must include among the symptoms of hydropericardium: great difficulty of breathing, general cyanosis, congestion of the jugulars, imperceptible or fluctuating, gurgling heart-beat, rapid and irregular pulse, dropsy, with great emaciation and feebleness.

*In pigs*, according to Rieck's observations, a rheumatic, fibrinous pericarditis often occurs. Out of 5,160 slaughtered pigs, the disease was found in 151, or in 3 per cent. It was sometimes complicated with pleuritis and peritonitis. Trasbôt and Anacker state that *in sheep*, an enzoötic pericarditis with very acute course occurs, which causes death in a few days.

**Differential Diagnosis.**—Confusion which occurs

with lung affections may be easily rectified by careful clinical examination, but this is more difficult with endocarditis and pleuritis. The differentiation of the former is based upon the character of the pericardiac sounds. This is (in contrast to the endocardiac after-sounds) a friction noise, which appears to arise close to the ear, and may sometimes be felt, and is not so strictly bound to the two phases of the heart's action, its systole and diastole, as are the endocardiac sounds. Moreover, one hears the heart-sounds quite normally along with pericardiac sound. Less easy is the distinction between pericarditis and pleuritis, which also is accompanied by characteristic rubbing noises. This, however, synchronises more with the rhythmical breathing than with beating of the heart, and in pleuritis the peculiar heart-phenomena are absent. Since, however, an internal pericarditis often occurs with an external one, which latter is synonymous with pleuritis, and since pericarditis, moreover, sometimes occurs secondarily after pleuritis, the distinction between the two is often very difficult or even impossible. Röll advises, in doubtful cases, to decide in favour of pleuritis, rather than pericarditis, owing to its more frequent occurrence.

**Therapeutics.**—The prognosis of inflammation of the pericardium is much more favourable in horses, dogs, etc., than in cattle, and its treatment therefore more successful. It consists in the avoidance of every excitement and movement, and the administration of cooling and antiphlogistic remedies. Among these come at the outset the local application of an ice-bag, or permanent irrigation of the cardiac region with cold water. Horses are best placed under a water-tap. To regulate the action of the heart, administer digitalis internally in small and repeated doses (horses 30 to 75 grains, dogs 1 to 1½ grains). Calomel, tartar emetic and the neutral salts may also be used. The external application of a mustard poultice, or of spirits of mustard on the cardiac region is also useful. Where the course is slow, try purgatives or diuretics. Should the exudation reach a dangerous point, or not diminish, despite all internal remedies, do not delay to puncture the pericardium in larger animals, and withdraw the exuded matter with a trocar. For the smaller animals, use Pravaz's syringe. In horses make the incision—after first cutting the skin—on the anterior edge of the fifth or sixth rib, a hand-breadth from the lower surface of the breast; in smaller animals on the site of

the dulness. To remove the danger of heart-failure or general weakness, administer stimulants : wine, camphor, coffee, alcohol, ether, etc.

**Hydropericardium.**—Dropsy of the pericardium is an accumulation of serous transudate in that organ. It is always a secondary condition which follows general engorgement of blood after diseases, such as chronic nephritis or hydremia. It may be clinically recognised by the disappearance of the heart-beat, enfeeblement of the heart-tones, with enlargement and diffusion of heart-dulness.

#### ACUTE ENDOCARDITIS.

**Etiology.**—Acute inflammation of the endocardium is most frequent in horses and cattle, also in dogs and pigs, but rarer in other domestic animals ; for instance, we have only known one case in a cat. Nevertheless, it is by no means so rare a condition as is generally thought. In most cases it is produced by infectious matter circulating in the blood. In cattle it is especially acute articular rheumatism (*polyarthritis rheumatica*) which not rarely becomes complicated with acute endocarditis (Meyer, Ruchte). In pigs erysipelas occurs as a frequent cause of endocarditis (Bang). We must also name septicæmia and pyæmia, e.g., in conjunction with septic metritis as well as general infectious diseases, such as foot-and-mouth. In pigs an acute articular rheumatism may even lead to death from acute endocarditis (Roth). In horses we find acute inflammation of the endocardium along with anthrax, pneumonia, rheumatic laminitis, and also with severe colic. In dogs there seems to be a contagious malignant endocarditis (Fröhner). Also, after intra-peritoneal injection of pneumococci cultures in a dog, an ulcerous endocarditis was observed (Michaëlis). Chills play an important part, along with infection, in producing so-called rheumatic or idiopathic endocarditis in horses (Trasböt). An inflammatory process may likewise spread to the endocardium from the myocardium and pericardium, and even from the lungs and pleura. Much more rarely is the cause traumatic. Ebinger saw a case of endocarditis in a cow produced by the blow of a horn with consequent broken rib. Finally, a predisposition to endocarditis seems to exist in old age, resulting from certain degenerative processes, such as arterial sclerosis of the valves of the aorta ; or in early youth from hereditary influences (Burke saw all the pups in a litter perish of the disease) ; and

also after recovery from a severe illness. Among drugs which, if administered too long or in too large doses, produce endocarditis, digitalis must take the first place.

**Bacteria.**—Wyssokowitsch has made some very interesting experimental investigations into the etiology of ulcerous endocarditis in animals. By irritation of the valves, with subsequent intra-venous injection of streptococcus pyogenes, he produced in rabbits an endocarditis resembling the ulcerous form (mycotic) in man. His experiments prove that micro-organisms which are otherwise harmless to animals may produce severe and fatal ulcerous endocarditis if the valves be injured, and that a very slight injury to the endocardium suffices for this end. He obtained similar results with staphylococcus pyogenes aureus and with coccus sepsis.—According to Dassy, every case of endocarditis is caused by micro-organisms. In addition to the pyogenic cocci, diplococcus lanceolatus capsulatus is also said to produce the disease.

**Post-mortem Lesions.**—These vary according to the degree and cause of the malady. In the slighter rheumatic forms we find small, knotty prominences, the size of a millet seed, especially on the free edges of the valves and on the tendons and papillary muscles. They are greyish-white to whitish-yellow in colour and, when present in numbers, form nodular, wart-like, lumpy protuberances, covered partly with bloody and partly with colourless and stratified fibrinous coagulæ (endocarditis verrucosa). These growths may develop on the margin of the valves to large polypous proliferations, which cause stenosis and insufficiency of the respective ostia and valves. If the granulæ are very small, the surface of the endocardium is rough and finely granulated. The endocardium is moreover diffusely swollen, opaque, reddened and covered with coagulæ; and circumscribed haemorrhagic foci are also found in the sub-endocardial and sub-epicardial tissue, as well as between the muscular fibres. The severe septic forms develop into necrosis and ulceration of the endocardium (endocarditis ulcerosa, maligna, diphtheritica, bacteritica). The ulcers are mostly local, their size from that of a lentil to a shilling, and are covered with dirty discoloured, friable, brittle and necrotic deposits, which are torn away by the current of blood and carried into other organs. Thus it is we find purulent metastases in the lungs, kidneys, liver, spleen, and even in the coronary arteries of the heart (abscess of the heart).

By means of the microscope we can prove that in verrucous endocarditis there is a sub-endocardial exudation

with small-cellular infiltration; and that there is also an exudation in the ulcerous form, but with coagulation and necrosis of the tissue and transformation into granular and flaky matter, with deposits of micrococci colonies. The former leads either to re-absorption of the exudation and recovery; or else, by metamorphosis of the white blood-corpuscles into new connective tissue, to thickening, shortening and adhesion of the valves and tendon-threads (insufficiency and stenosis, valvular defects, chronic endocarditis). Sometimes, by detachment of the adherent growths of thrombi, it leads to benign stoppages in the kidneys, brain, etc., which mostly scar over. The ulcerous form, on the contrary, ends in a complete necrosis of the tissue, which is more or less diphtheritically infiltrated.

As regards the distribution of endocarditis upon the two halves of the heart, the proportion is obviously different in domestic animals from what it is in man. Whereas in man it usually attacks the left heart, in animals the right side is by no means rarely affected; indeed it would almost seem to suffer more frequently than the other. Thus, according to Meyer, in nine cases of endocarditis the tricuspid valves were eight times implicated, and the bicuspid only four, the valves of the aorta being affected in two cases. Particularly among cattle do we find endocarditis of the right side, whereas among men this is extremely rare and mostly of foetal origin.

**Symptoms.**—Acute endocarditis reveals itself first in tempestuous action of the heart, with palpitating, irregular and, later, diffused beat. The number of heart-beats sometimes exceeds that of the pulse (Trasbôt), so that we find, for instance, that in horses the proportion will be as 160 to 70. The action of the heart may, in fact, be so vehement that the whole body is shaken with every stroke. The pulse is also rapid, and its speed may be between 80 and 160 a minute in horses and cattle, and in dogs may vary from 120 to 240. It is irregular, frequently intermittent and very weak, indeed not rarely imperceptible. The heart-sounds are at first normal, but soon become abnormal, the first one in particular being very dull and sometimes amalgamating with the second to form only one sound. Later, certain characteristic endocardiac after-sounds often arise, above all a blowing, contracted (systolic) noise with the first heart-sound, and more rarely—though of greater pathognostic importance—certain prolonged (diastolic) rustling and vibrating sounds. In acute endocarditis the tem-

perature is always raised to fever point, and is especially high in the septic form. Trasbôt found in horses a temperature of  $104^{\circ}$  and  $106^{\circ}$  F.

Breathing is often so extremely laboured and accelerated that one might at first take the disease for pneumonia or pleuritis, and in some cases it rises to decided dyspnoea. The general condition of the sick animal is very bad; along with serious brain affection, one observes great lassitude and weakness as well as periodical fits of colic. To these come symptoms of metastasis in the lungs (attacks of choking), in the kidneys (bloody urine), in the extremities (lameness) and in the brain (apoplectic fits). In the septic form a general hæmorrhagic diathesis is developed.

**Course.**—Sometimes this is very acute, leading to death in a few hours or days; at other times the malady endures for weeks, and an acute endocarditis may eventually develop into the chronic type. In the latter case acute inflammatory attacks readily recur. The result is always different according to whether we have a benign, verrucose, or severe, septic, ulcerous endocarditis to deal with. In the latter a fatal ending through development of a general septic infection, or of metastatic foci in the lungs, kidneys or brain, is much more frequent than with the verrucose form, which, nevertheless, leaves behind it serious changes in the shape of valvular defects.

**Differential Diagnosis.**—Acute endocarditis can, when its course is very acute, be confounded with anthrax, and also with septicaemia, inflammation of the brain, petechial fever, etc. And, on the other hand, owing to the extreme difficulty of breathing, it is not easy to distinguish from incipient pneumonia, from congestion of the lungs and oedema of the lungs. But yet more difficult is a differential diagnosis between endocarditis and myocarditis. In all these cases one must rely on thorough examination of the heart. Without the after-sounds, a diagnosis of endocarditis is impossible. But we must remember that systolic after-sounds especially occur also in anaemia and leucæmia (so-called inorganic murmurs), whereby the diagnosis of endocarditis is rendered still more uncertain.

**Therapeutics.**—The treatment of acute endocarditis consists in avoidance of all excitement or movement, regulation of the action of the heart by digitalis, reduction of the fever by

salicylate of soda, tartar emetic, antipyrin and other fever remedies. In small animals an ice-bag can be laid upon the region of the heart, and in larger ones the same part can be permanently irrigated with cold water. The efficacy of derivative inunctions is a disputed point. If strength fail rapidly, use camphor, ether, spirits of wine, atropine, hyosyamine, caffeine, etc.

#### CHRONIC ENDOCARDITIS. VALVULAR DEFECTS.

**Etiology.**—Valvular defects are extraordinarily common among our domestic animals, especially in dogs, horses and pigs. In by far the larger number of cases they are traceable to organic changes in the valves produced by chronic endocarditis. Such chronic forms most frequently develop from an acute endocarditis. Thus, for instance, many valvular defects in cattle spring from a previous acute articular rheumatism with simultaneous acute endocarditis. In horses such defects have been observed after acute infectious disease, such as influenza; and in pigs as a complication and sequel of erysipelas. Apart from this acute development, there is also a creeping, chronic endocarditis, which is provoked by repeated chill, over-exertion or excitement. Indeed in pigs chronic endocarditis occurs not seldom without any evident erysipelas. Arterio-sclerotic processes at the root of the vessels of the heart will lead to chronic changes in the valves affected, particularly in old age. Finally, hereditary pre-disposition or ante-natal developments must be taken as the causes of many valvular defects.

A minority of defects in the valves do not arise from endocardiac processes, but from congenital faults or new growths in the heart. Thus such inborn defects occur in the septum of both ventricles, and indeed the septum may be absent entirely in the cow. Behrens noticed a perforation of the mitral valves in a horse, which was accompanied by a systolic after-sound and led eventually to hypertrophy and dilatation of the heart. In a dog which died suddenly, Jungers found a polypous new growth with long stalk, which firmly closed the left auriculo-ventricular orifice. Hink discovered, as the cause of sudden death in a cow, a fibro-sarcoma which had been detached from the wall of the left ventricle and completely stopped up the aorta. Franck saw an imperfect stoppage of the foramen ovale in a dog which suffered from epileptiform fits and general cyanosis. New growths in the heart or its vicinity, such as sarcomata, carcinomata, fibroids of the valves, myxomata or ulcers in the bronchial glands, may lead to stenosis of the valves of the heart.

But as the last-named conditions are of rare occurrence, we shall here only treat of such valvular defects as are caused by chronic endocarditis.

**Post-mortem Lesions.**—Changes in the valves produce either inability to shut (insufficiency) or narrowing (stenosis).

1. The *insufficiency* of a valve arises from shrinkage, as well as shortening of its tendinous fibres. In the course of chronic endocarditis a change in the connective tissue of the valves occurs, with shrinking and atrophy, nay, even complete disappearance. Moreover, owing to the simultaneous shortening of their tendinous fibres, the valves cannot distend to their full size. The orifice is thus enlarged, and even when the valve is closed, an open cleft is left, through which the blood is driven back again to the other side of the valve. Perfect or independent insufficiency or stenosis is rare, as the endocardiac process mostly leads both to inability to shut and to contraction. We therefore generally find stenosis and insufficiency side by side (combined valvular defects). Secondary anatomical changes in the body produced by valvular defects in consequence of disturbed circulation are: hypertrophy and dilatation of the heart; engorgement of the liver, brown induration of the lungs, engorgement of the kidneys and of the spleen; dropsies, especially ascites, hydro-thorax, hydropericardium, anasarca; emaciation; and diffuse haemorrhage.

2. *Contraction of the Orifices* is produced by the thickening of the valves and also by proliferation of the endocardium. The lumen of the orifice is found to be narrowed by polypous, wart cauliflower-like growths from the valves, or almost filled by tumour-like masses. The edges of the valves are thickened and spongy, sometimes covered with chalky incrustation, and the several valves are not rarely grown together at their free edges. They have thereby lost their free mobility as well as their power of full distention, and are changed rather into rigid, unyielding, narrow rings, which at times are as hard as cartilage or bone. Consequently the normal quantity of blood cannot pass through them in a certain time and must therefore be forcibly driven through.

**Occurrence.**—These valvular defects are very common among our domestic animals, occurring most frequently in aged dogs and horses, also in pigs. A very large number of draught-horses and draught-dogs are incapacitated by them. But the literature on valvular defects in cattle is also abundant,

a result of the frequency of acute articular rheumatism among them. Such defects have also been noted in birds. The distribution of the lesions between the two halves of the heart in domestic animals is pretty even. Among 37 cases of endocarditis described in veterinary literature, 20 were in the left and 17 in the right heart. These were further sub-divided : 14 affected the mitral, 6 the valves of the aorta, 15 the tricuspid and 2 the valves of the pulmonary-arteries. The tricuspid valves seem therefore to have suffered a little oftener than those of the mitral, a proportion which holds good, especially in cattle. Among 7 cases in cattle, 6 were on the right side, and 1 was on both ; none on the left heart alone. In horses the proportions seem to be about the same as in man : of 15 cases, 7 were on the left side, 5 on both sides, and 3 on the right.

That valvular defects are of extremely frequent occurrence, not only in dogs, but also in horses, is proved by a summary prepared by Nocard. In 42 horses bought for purposes of operation, he found localised changes at the root of the aorta in 38, and in 4 others chronic endocarditis on the crescent-shaped valves of the aorta, and also upon the mitral valves. They were all old horses. The intensity of the changes varied in individuals, the diseased valves being in some merely thick and stiff, in others drawn as by a cicatrix, and covered with fibrous vegetation, and again they were corroded, denticulated and perforated in many ways. The changes had been diagnosed during life. In all the animals there was a hissing sound, which began with diastolic murmur, and ceased with the first heart-sound. This sound varied in intensity and timbre. Cadiot and Ries found among 14 cases of valve-defects in horses that the aortic valves were affected in 10, the mitral-valve in 1, and that both were affected in the other 3. Among 13 cases in dogs, 5 were on the mitral, 4 on mitral and tricuspid, 2 on the latter alone, 1 on the aorta and 1 on aorta and mitral.

**General Symptoms of Valvular Defects.**—Both stenosis and valvular insufficiency cause an engorgement of the blood-stream. Defects in the mitral cause engorgement in the pulmonary veins and lungs ; in the tricuspid the engorgement is in the systemic veins. If the aortic or pulmonary valves be affected there will be an engorgement of the corresponding ventricle. These disturbances of circulation are at first equalised by increased activity on the part of the heart, i.e., of the divisions concerned, so-called *compensatory hypertrophy of the heart*. A defect of the heart thus compensated may exist for years without any serious morbid symptoms appearing, and it is only after severe exertion or excitement that any marked disturbances of circulation can be perceived. But in

process of time loss of strength and weariness of the muscles of the heart show themselves, these being no longer able to cope with their task; whereupon pronounced indications of engorgement of the lungs, and also of the vena cava, make their appearance. Only in this stage do the patients come under observation and treatment, and the aspect of the malady is in the main the same then as in the later stage of hypertrophy of the heart. The symptoms of a decreasing heart-compensation are:

1. *Acceleration of pulse and heart-beat*, easily excited activity of the heart, irregularity of pulse and heart-beat, palpitation. In isolated cases we find the contrary with the pulse, viz., diminished frequency (Bradycardie).
2. *Difficulty of breathing*, and after much movement dyspnoëic attacks. This shortness of breath, which becomes a prominent symptom of the complaint, may be explained by engorgement and slower circulation of blood in the lungs, with subsequent permanent changes in that organ (brown induration of the lungs, contraction of the lumen of the pulmonary alveoli by the dilated lung capillaries).
3. *Increasing weakness, general emaciation*, slight sweating after brief exertion, and all this without any fever. With draught animals, conspicuously rapid exhaustion during work is one of the earliest signs of incipient weakness of heart.
4. *Attacks of dizziness*, particularly after severe exertion (frequent in draught-dogs).
5. *Cyanosis* of the visible mucous membranes, in pigs also of the entire skin, particularly on the ears and back; great distension of the visible veins (those on the jaws, jugular veins); venous pulse.
6. *Hydrops of the bodily cavities* (ascites, hydrothorax, hydropericardium), as well as of the subcutaneous connective tissue (œdema on belly, evacuatory organs, chest and legs).
7. *Albuminuria and diminished secretion of urine*, being a sign of engorgement of blood in the kidneys.
8. Sometimes a *jaundiced discolouration* following upon engorgement fever.
9. *Embolii in peripheral organs*, arising from emboli in the heart, also in the lungs, brain, kidneys, liver, spleen and extremities (lameness, swelling of the joints).
10. Of pathognostic importance for the symptomatology of valvular defects are, finally, the *abnormal sounds, perceived on auscultation of the heart*, which will be more fully described later.

**Symptoms of the Several Valvular Defects.**—The most important of such defects are the stenosis and insufficiency of the mitral valves (horses and dogs) and tricuspid valves (cattle), also of the aortic valves (horses); much less common are defects in the pulmonary.

#### I.—INSUFFICIENCY OF THE MITRAL VALVE.

This, commonest of all valvular defects in horses and dogs, causes a back-flow of blood during the systole through the unclosed orifice into the left auricle, whereby the whirling motion of the retreating blood, as well as its contact with the venous blood, produces a louder or softer blowing, hissing or whirring after-sound, which is sharply systolic. The left auricle being fed from two sides, and therefore very full, becomes dilatated and hypertrophic. The discharge of blood from the lung-veins is hindered, and an engorgement takes place in these veins, in the lung-capillaries, and even in the lung-arteries, and consequently also in the right auricle and right ventricle, bringing in its train hypertrophy of the heart. As a result of the high pressure existing in the pulmonary artery, the second heart-tone (pulmonary tone) grows louder, more flapping, as it is usually expressed, and accentuated. Should compensation on the part of the right heart decrease, then cyanosis, dyspnoea, congestion of the jugulars, venous pulse, hydrops, etc., etc., set in:

*Characteristic symptoms* of mitral insufficiency are: systolic after-sounds, abnormally strong diastolic pulmonary tone, hypertrophy of the right side of the heart, feeble pulse, shortness of breath, engorgement of the venous system.

#### II.—STENOSIS OF THE ORIFICE OF THE MITRAL VALVES.

In contraction of the left auriculo-ventricular opening during diastole blood cannot pass unchecked from the auricle into the ventricle, but is only partially forced through the narrow opening with a whirling motion. Thus a diastolic (pre-systolic) after-tone is produced. Moreover, the left ventricle is only partly filled and shrunken, and the pulse very small and feeble. The left auricle is dilated and hypertrophied, engorgement sets in throughout the pulmonary system, leading to hypertrophy and dilatation of the right heart; in consequence of which the pulmonary tone is much accentuated.

*Characteristic symptoms* of mitral stenosis are: diastolic after-sound, loud pulmonary tone, very small pulse, very grave signs of engorgement, especially on the part of the lungs.

### III.—INSUFFICIENCY OF THE TRICUSPID.

Inability of the right auriculo-ventricular valve to shut brings on a regurgitation of blood from the right ventricle into the right auricle, combined with a systolic after-sound. The backward movement of the blood in the auricle is carried on into the veins and causes a venous pulse, with engorgement in the venous system (cyanosis of the visible mucous membranes). The right auricle becomes dilatated and hypertrophied, and later the whole heart becomes distended. On relaxation of the latter, engorgement of blood in the lungs takes place, with formation of thrombi therein (dyspnoea), as well as in the *venæ cavæ* (hydrops).

*Characteristic symptoms* of tricuspid insufficiency are: systolic after-sound, venous pulse.

### IV.—STENOSIS OF THE ORIFICE OF THE TRICUSPID.

This, which is rare in man, though frequent in cattle, is recognised by a diastolic after-sound, great repletion, dilatation and hypertrophy of the right auricle, venous pulse, retardation of the blood-stream in the pulmonary artery with subsequent formation of thrombi, and embolic conveyance of the thrombi into the lungs; hypertrophy, and also enlargement of the right ventricle, disturbance of breathing, and symptoms of dropsy.

### V.—INSUFFICIENCY OF THE AORTIC VALVES,

which is rather common in horses, causes a back-flow of blood during the diastole from the aorta into the left ventricle. Since a part of the blood therefore rushes back and meets that which issues from the left auricle, a diastolic secondary or after-sound is produced, which may also be felt as a whirring sensation. The overcharged condition of the left ventricle produces extreme dilatation and hypertrophy of it. The hypertrophic state of the left heart causes also a strong arterial pulse, the curve of which, however, sinks abruptly owing to the retreat

of a portion of the blood into the left ventricle : pulsus celer, i.e., a rapid, jumping pulse. The diastolic aorta-sound is sometimes audible even in the carotid.

*Characteristic symptoms* of insufficiency of the aorta are : buzzing, diastolic after-sounds, extreme dilatation and hypertrophy of the left heart, strong arterial pulse, pulsus celer and arterial sounds.

#### VI.—STENOSIS OF THE ORIFICE OF THE AORTIC-VALVES

sets the blood which streams through the narrowed valves into whirling movement, producing thus a systolic after-sound. The pulse is at the same time made slower and very small, and the left ventricle becomes hypertrophic. In extreme stenosis, symptoms of anaemia of the brain, vertigo, etc., appear.

*Characteristic signs* of aorta-stenosis are therefore : systolic after-sound, slow and very small pulse, hypertrophy of the left side of the heart, cerebral anaemia, etc.

#### VII.—INSUFFICIENCY OF THE PULMONARY VALVES

is very rare, and gives rise to a diastolic after-sound, with hypertrophy and dilatation of the right ventricle, and, on decrease of the activity of the heart, to extreme disturbance of respiration and circulation.

#### VIII.—STENOSIS OF THE ORIFICE OF THE PULMONARY VALVES

produces a systolic after-sound, hypertrophy of the right side of the heart, engorgement in the venous system, and very soon difficulty of breathing, owing to disturbed compensation of the heart.

**Diagnosis of Valvular Defects.**—The recognition of a valvular defect is for several reasons not always easy. First the after-sounds, both systolic and diastolic and without any organic change in the valves, occur also in leucæmia and anaemia as so-called un-organic heart-tones. And on the other hand, these after-sounds may be lacking, especially at the beginning, even where there is a genuine valvular fault, and thus the most important diagnostic factor be missing. Furthermore, two or more valvular defects may exist side by side in every possible combination, whereby the symptoms of the one may be masked by those of the other. Thus, stenosis

and insufficiency are most frequently found together. Finally, in the last stage, viz., that of incipient disturbance of compensation, the symptoms of all are so very much alike (disturbed circulation in the lung and venous systems) that differential diagnosis is rendered extremely difficult, and it is unfortunately only in this stage that attention is first called to the ailment. Yet it is true that frequently one valvular defect so far predominates over the rest in its symptoms as to enable it to be at least approximately diagnosed. In order therefore to furnish a few points on which to base a differential diagnosis, we would beg to submit the following as marking the several defects :

1. A *systolic after-sound* is noticeable in mitral insufficiency, tricuspid insufficiency, aortic stenosis and pulmonary stenosis. The first valvular defects are rather frequent, the last one is scarcely ever observed. Insufficiency of the tricuspid occurs most frequently in cattle and is associated with a venous pulse. Mitral insufficiency is found more in horses and dogs, and is accompanied by a weak pulse ; stenosis of the aorta with a very small and at times retarded pulse.

2. A *diastolic (pre-systolic) sound* may arise from stenosis of the mitral or tricuspid, and from insufficiency of the aorta or pulmonary. The latter is so rare as to be hardly worth consideration. Tricuspid stenosis is most common in cattle, and is accompanied by a venous pulse and severe dyspncea, caused by formation of thrombi in the right heart. Mitral stenosis may be distinguished from aortic insufficiency by the small and irregular pulse, which in the latter is very strong and tends to become rapid. The diastolic sound in horses and dogs is most frequently traceable to insufficiency of the aorta.

**Therapeutics of Valvular Defects.**—A compensated valvular defect requires no treatment ; one must merely feed the animals well, and guard them against over-exertion and excitement. Not until the hypertrophied heart begins to relax in its activity, and disturbances of circulation are thereby set up in the lungs and venous system, must recourse be had to such remedies as may regulate the heart, reduce the excessive speed of the pulse, and increase the blood-pressure in the arterial system, thus giving the heart the pauses necessary for recuperation and reception of nutriment. For such purposes digitalis is a very valuable drug ; it equalises the disturbed circulation, raises the arterial blood pressure and lessens the

frequency of the pulse. It is given either as powder, or as an infusion of one-third strength, several days in succession. Its operation must, however, be carefully watched, and its use then discontinued for a while. The doses must be small. For example, we give a dog an infusion of 15 to 30 grains fol. digitalis in eight ounces of water, daily one to two tablespoonfuls (for small dogs say teaspoonfuls); horses, 30 to 75 grains; cattle, 1 to 2 drams pulv. fol. digitalis upon bread, or in saline remedies. Besides digitalis, caffeine is to be recommended when much dropsy is present, it having the advantage of not being poisonous when administered for a considerable length of time, and possessing moreover a stronger diuretic effect. Give it in the form of its salts, e.g., caffeine sodio-benzoate, to dogs daily 4 to 15 grains, horses and cattle 1 to 2½ drams. Tincture of strophanthus acts similarly (horses and cattle 2½ to 6 drams, dogs 10 to 25 drops); also diuretin (dogs every two hours 4 to 8 grains). Iodide of potassium is highly recommended as an adjuvant to digitalis (horses 1 to 2½ drams).

From a symptomatic point of view, puncture is advisable where there is excessive dropsy in the cavities (ascites, hydrothorax, hydropericardium); also a diuretic treatment (the above-named heart-specifics, diuretin, scilla, acetate of potash), and a diaphoretic or saliva-stimulating course (pilocarpine, arecoline). Drastics may also procure temporary relief. To combat general weakness, stimulants should be used, and against abnormal palpitation, narcotics, especially morphia in subcutaneous injection (horse 8 grains, dog ¼ to ½ grains).

#### MYOCARDITIS.—INFLAMMATION OF THE MUSCLES OF THE HEART.

**Etiology.**—The causes of myocarditis are occasionally chills or over-exertion, but principally secondary infection, as from anthrax, foot-and-mouth disease, septicæmia, pyæmias, tuberculosis, etc. Furthermore, an inflammation of the endocardium or pericardium may extend to the myocardium. Injury to the heart, which is very common in cattle, though always complicated with pericarditis (see earlier in this chapter), may also produce myocarditis, as also in horses may be done by shock or contusion of the muscles of the heart.

**Post-mortem Lesions.**—The changes in the myocardium are extraordinarily varied, according as a parenchymatous, an interstitial, an indurative or an abscess-forming myo-

carditis be present. Whereas in interstitial and abscess-forming myocarditis the inflammation is usually focal, circumscribed and even disseminated, in the parenchymatous form it generally occurs diffusely.

1. *Acute parenchymatous myocarditis* is rarely found as an independent and primary disease. The inflammation attacks not only the muscular fibres, but is complicated with interstitial infiltration. Viewed in the mass, the muscles show a spotty discolouration, and appear from grey-red to greyish-yellow, chalky and even whitish yellow, of tender consistence, and their fibres seem obliterated, indistinct and homogeneous. The microscope shows a loss of the correct striation, as well as a dusky opacity in the muscular fibres, along with considerable small-cellular infiltration in the interstitial connective tissue, the muscles being thrust apart by a layer of white blood-corpuscles.

2. *Chronic indurated myocarditis* appears in the form of whitish, shining, so-called callosities of the heart, which are like connective tissue in texture, and sometimes as hard as cartilage, also of tendinous deposits (milky and tendinous spots, macula lactea). These vary in size and penetrate the muscles of the heart, especially the left near the apex, being found sunk beneath its sectional surface, and occasionally making its walls so thin that they cause the exterior to bulge (aneurysm of the heart). We also find the fibrils of the muscles sometimes fattily-degenerated, granular and surrounded by very abundant growth of connective tissue. The whole develops from a small-cellular infiltration of the interstitial connective tissue, with induration and cicatrisation, and leads usually to secondary hypertrophy of the heart.

3. *Purulent myocarditis, or abscess of the heart*, occurs chiefly in pyæmia:—in cattle with puerperal metritis, in foals with omphalo-phlebitis. The abscesses are either milinary and disseminated through the cellular connective tissue; or larger, varying from a hazel to a chestnut in size, appearing in conjunction with an embolic stoppage (metastatic myocarditis). Abscesses of the heart have been observed in both cattle and horses; in the latter they sometimes become caseous and even show a chalky coating (ossification of the heart).

Closely related to myocarditis, and to some extent resembling its first stage, although much commoner, is the degeneration of the muscles of the heart muscles occurring in the course of infectious diseases:—opaque swelling, fatty degeneration, etc., whereby the heart as a

whole appears very tender and flabby, as though boiled, and is also discoloured. But, on the other hand, there is no small-cellular infiltration. The clinical aspect of the complaint is very similar to that of myocarditis, and both of them produce insufficiency of the muscles of the heart. The so-called callosities of the heart are not always developed upon an inflammatory base, but arise, according to Ziegler, much more frequently from a myomalacia, *i.e.*, from softening of the myocardium, following on anaemic necrosis, a condition which has hitherto been erroneously termed parenchymatous myocarditis. This myomalacia cordis develops from a haemorrhagic stoppage caused by arterio-sclerotic or embolic processes, with which are complicated, one after the other, arterial anaemia, softening, caseation, resorption of the waste matter with final cicatrisation. Just in the same way scabs of connective tissue form on circumscribed foci of softening in the brain, or in stoppages of the kidneys.

**Symptoms.**—Are those of disturbance of function of the cardiac muscles, *i.e.*, of insufficiency or weakness of the heart, and correspond with the symptoms of dilatation of that organ. The heart-beat is at first very tumultuous, throbbing, extended, and at times is both visible and audible at some distance; it is also irregular; later it grows very feeble. The pulse is very rapid, small, irregular and often imperceptible, counting sometimes in horses continuously 80 to 120 strokes a minute. Respiration grows difficult and rapid. Then comes great weakness and prostration, trembling, fits of dizziness and great excitability. Temperature is raised in accordance with the generating cause. In cases of long duration, general appearances of engorgement set in, as with hypertrophy of the heart. In other instances the inflammation of the myocardium takes a very acute course, and the animals suddenly and apoplectically collapse. In this way many cases of foot-and-mouth or of tuberculosis end quite unexpectedly. Death results from paralysis or rupture of the heart. Both acute parenchymatous and suppurative myocarditis commonly end in this way.

**Differential Diagnosis.**—The differentiation of primary myocarditis from other heart-affections, and especially from passive enlargement, endocarditis and pericarditis, is very difficult, indeed often impossible, as the symptoms are very similar. For these reasons a diagnosis is especially difficult whenever the characteristic signs of pericarditis, endocarditis or a valvular defect are not present. In general the probabilities would favour a diagnosis of myocarditis when severe phenomena of heart-insufficiency are noted without any demonstrable

organic change (after-sounds, rubbing noises). The identification of a secondary myocarditis is far easier.

**Therapeutics.**—The treatment for inflammation of the heart corresponds with that for weakness of the same organ, it consists therefore in avoidance of all excitement or exertion, strengthening the muscles by nourishing food, stimulants, digitalis, caffein, strophanthus, camphor, atropine, hyoscyamine and alcohol, as well as in administration of febrifuges. In cases with very acute course no treatment is of any avail.

**New Growth in the Muscles of the Heart.**—Among these, sarcomata seem to be of special clinical importance. In a dog which died with symptoms of weak heart, dissection revealed grey, circumscribed foci in the myocardium, which were found under the microscope to be a sarcomatous infiltration of the fleshy part of the heart, with atrophy of the muscular fibres. At the same time there were multiple, metastatic, sarcomatous foci in the kidneys (Fröhner).

#### HYPERTROPHY AND DILATATION OF THE HEART.

**General Observations.**—Hypertrophy and dilatation of the heart in their advanced stages are anatomically and clinically distinct, and ought to be discussed in different chapters. But in practice they can only be differentiated with difficulty, because they very often occur together, viz., along with valvular defects; or, after having for a short time existed independently, speedily pass one into the other; and also because in the later stages of disease a differential diagnosis between them is usually quite impossible, owing to the similarity of their symptoms. The relationship of the two is further expressed in our terminology, which describes hypertrophy of the heart as active, and dilatation as *passive enlargement of the heart*.

**Anatomical Notes.**—Both hypertrophy and dilatation cause an enlargement of the heart, but in the former this increase of volume takes the form of a growth in the substance of the cardiac walls, and the second in enlargement of its hollow cavity.

i. The enlargement of the heart in *hypertrophy* can only be produced by a thickening of its walls; and we call this somewhat rare form a simple hypertrophy of the heart. But in by far the greater number of cases this increase of thickness is accompanied by an enlargement of the cavity, so-called

'eccentric hypertrophy or active enlargement of the heart, and this is usually what is meant when we speak of hypertrophy of the heart. Whether a third form exists in which the cavity is diminished while the walls are thickened, or a so-called concentric hypertrophy of the heart, is doubtful. Under the name of false or spurious hypertrophy we understand an enlargement caused by foreign deposits within the cardiac walls. True hypertrophy of the heart may be either total or partial, viz., affecting only one ventricle, or it may be circumscribed and confined to only one papillary muscle. The two latter forms are most common. The left heart is by far most frequently hypertrophied. The shape of the heart then becomes elongated and cylindrical, whereas in right-hand hypertrophy it is more flattened and broadened, while a totally hypertrophied heart is almost round and often attains colossal dimensions. Thus Stephenson found a horse's heart weighing 32 pounds, and Gerlach one of 19 pounds; Herran measured the heart of an ox which was 14½ inches long and weighed 36 pounds, the walls of which were incrusted with lime-salts. The wall of the left heart is not unfrequently more than twice or thrice its usual thickness. Its structure may nevertheless remain quite normal, although hypertrophied. But, as a rule, the muscles are harder, of tougher, firmer consistency, sometimes penetrated by proliferations of interstitial connective tissue, being also darker (brownish-red in colour), and may later be marked with lighter spots and stripes (deposits of pigment; fatty degeneration of the muscular fibres). The wall of the right heart when hypertrophied gapes if cut open, instead of collapsing.

2. *Simple dilatation of the heart* produces enlargement essentially by distention of its cavities, by which its walls must naturally become weaker and thinner. Relaxed and distended walls are characteristic of passive enlargement of the heart, and whereas in the active form the heart gains in strength, in the passive it loses it. Dilatation of heart is usually found on the right side. In dilatation of the left side the walls collapse on being cut, instead of gaping. The structure of the myocardium may be normal, but the wall is usually relaxed, anaemic, fattily-degenerated, friable and easily torn, of pale, yellowish-brown colour and very thin, indeed in some places quite transparent, consisting only of endocardium and epicardium. Along with these appearances a bulging-out of the wall may also be developed, so-called aneurysm of the heart. In extreme cases of dilatation the heart may be more than double its usual size.

**Etiology.**—The causes of eccentric hypertrophy of the heart are usually to be sought in circumstances which offer resistance to the circulation and thus increase the blood-pressure. Among them we must mention :—

1. *Increased physical exertion*, so-called *labour hypertrophy* or *idiopathic hypertrophy* of the heart, which occurs particularly in race-horses as a result of very severe—and though only brief, yet often repeated—exertion ; also in draught oxen and hunting hounds. This is the reason why a race-horse's heart is larger than that of less noble breeds. Thus, for instance, the heart of the English pure-bred stallion Helenus weighed  $13\frac{1}{2}$  pounds. The development of idiopathic hypertrophy of the heart has been explained by assuming that the contraction of the muscles of the trunk during hard work leads to contraction also of the muscular arteries, and thus to an increased pressure of blood, which necessitates greater activity on the part of the heart, causing a muscular hypertrophy.

2. The various *obstacles to circulation in the blood-vessels* cause secondarily a so-called *symptomatic hypertrophy* of the heart. Under this head belong aortic aneurysms, congenital stenosis of the aorta, thrombi, compression of the aorta by new growths, atheromatous and arterio-sclerotic processes, excess of blood (plethora).

3. *Valvular defects* of the heart lead frequently to hypertrophy of that organ, owing to the disturbance they cause in the circulation ; this is a so-called *compensatory hypertrophy*, because such disturbances are supposed to be balanced by it.

4. *Lung-diseases*, such as emphysema of the lungs, compression by exudations or transudations, adhesion to the costal walls, interstitial glandrous or tuberculous pneumonic processes, which shut off part of the pulmonary vessels from circulation, increase the blood-pressure in the region of the right heart, and thus give rise to a hypertrophied heart.

5. *Adhesion of the heart to the pericardium* also increases the demands upon the activity of the heart by hindering its free action, and thus causes it to grow in size.

6. Finally, *chronic inflammation of the kidneys* is almost always complicated with hypertrophy of the heart.

*Passive enlargement of the heart*, or *dilatation* of the same, forms usually the continuing stage of the active form, in so far that the hypertrophic heart is finally unable to meet the demands made upon it, its action grows weaker, and it then becomes distended by the abnormally increased blood-pressure.

In this way fatty-degeneration of the heart, myocarditis, endocarditis, sclerosis of the coronary arteries, changes wrought in the myocardium by high fever, infectious diseases, poison and anaemia all produce dilatation. Finally, a passive enlargement of the heart may develop acutely after acute lung-affections with serious interruption of circulation and extreme increase of blood-pressure in the region of the right heart. For instance, in acute pneumonia of horses, if long continued, an incipient hypertrophy of the heart may arise, which, however, disappears later. On the contrary, chronic changes in the lungs always relieve a hypertrophy of the heart, as has been already stated, unless the animals be too feeble ; in the latter case, dilatation nevertheless occurs in spite of the chronic course, owing to the lack of sufficient nutritive material.

**Symptoms.**—A genuine, and especially a compensatory, hypertrophy of the heart usually develops so gradually that it may exist for years without any visible disturbance of general condition. In extreme cases, as, for instance, during chronic nephritis, it reveals itself by a full pulse, exceedingly powerful heart-beat, very loud and clear heart-sounds and in extension of the cardiac dulness. This, which is the sole characteristic symptom of "hyperkinese," of hypertrophy of the heart, does not, however, make its appearance very often ; for one reason, because the hypertrophy sooner or later passes into dilatation and exhaustion, owing to gradually increasing insufficiency in the action of the heart, and for another, because an idiopathic hypertrophy, which is not produced by other organic changes nor complicated therewith, is of rare occurrence. Hence it comes to pass that the cases of ill'ness designated by the name of "Hypertrophy of the Heart" in literature and in practice mostly refer to complications of active and passive enlargement of the organ, indeed often to the latter alone. The morbid symptoms of this double affection, which are exactly those of non-compensated valvular defects, differ considerably from those of the true hypertrophy of the heart as named above.

The symptom which is common to both active and passive enlargement of the heart is extension of cardiac dulness as demonstrable by percussion. With great dilatation, e.g., in dogs, this may reach to the region of the last false ribs. Incipient exhaustion of the hypertrophied heart then shows itself in oppressed breathing, palpitation, a quite abnormal pulse, hyperæmia and anaemia of the brain, fits of dizziness, as well as in

extensive disturbances of circulation with their consequences. The most prominent sign is the strong throbbing of the heart, which can be felt on both sides, and is even visible from a distance and shakes the whole body. The heart-sounds are irregular, the first very loud, with metallic ring and sometimes vibrating, and the second mostly weak or even quite absent. The pulse, in marked disunison with the heart, is generally weak or even quite imperceptible (hypokinesis); after very slight exertion extremely rapid, irregular and uneven. Not unfrequently a venous pulse can be observed in the jugular vein. Even when at rest, breathing is quickened, and rises to dyspnoea if the animal be used (so-called cardiac asthma, pursiness), or even to suffocation. The animal staggers, falls to the ground and dies of apoplexy. The later symptoms occur sometimes in the form of fits of dizziness, trembling, twitching, sweating and dyspnoeic, rattling breath.

The interruptions which occur later on in circulation soon affect the brain (congestion and anaemia of the brain in the form of vertigo and fainting), also the bronchial membrane (bronchitis), the digestive tract (chronic intestinal catarrh), the liver (engorgement of the same), and the kidneys (engorgement with albuminuria and oliguria). A marantic thrombosis of the arteries (paralysis of the hind-legs caused by thrombosis in the region of the posterior aorta) is also a not uncommon sequel to disturbed circulation. Lastly, general cyanosis and dropsy develop (ascites, hydro-pericardium, hydro-thorax, anasarca); the lower breast, lower belly and extremities swell, the animal grows very thin and either perishes of marasmus or of embolism of the lungs, caused by coagulation of blood in the heart, paralysis of the heart, haemorrhage on the brain and carbonic-acid poisoning.

The symptoms just described are commonest in old dogs, which are brought to be treated for great shortness of breath, or for frequent dizziness, also in horses when examined for asthma or pursiness; but they also occur in other animals. Thus Herran reports on a case of hypertrophied heart in an ox, which gradually wasted away with the appearances of chronic hard-breathing, and was obliged constantly to rest after every exertion owing to tumultuous heart-throbbings.

**Differential Diagnosis.**—Hypertrophy and dilatation of the heart are often mistaken for a primary lung affection, e.g., congestion of the lungs or pneumonia. Owners are par-

ticularly liable to be misled to this idea by the great prominence of the symptom of difficult breathing. But a careful examination of the lungs by auscultation and percussion, the gradual appearance and long persistence of the condition, as well as a positive demonstration of some heart-affection enable one soon to learn the truth. Active and passive enlargement of the heart differ moreover from primary palpitation by absence of extended cardiac dulness, as well as of the grave consequences of the latter. The palpitation also is not continuous but intermittent. More difficult is the differentiation from hydro-pericardium and myocarditis, especially the chronic form of the latter, which is accompanied by induration ; indeed it is often impossible, as the symptoms of chronic myocarditis are precisely those of hypertrophy of the heart. On the other hand, valvular defects may be differentiated from the latter with ease, as they exhibit quite characteristic heart-sounds, without which they could not be diagnosed.

**Therapeutics.**—The chief aim in prescribing for hypertrophy and dilatation of the heart is to find out their causes and remove or combat them. We must, however, at once observe that any hindrance of a compensatory hypertrophy which serves to balance existing defects is objectionable, especially the use of digitalis. In this respect the nature of the pulse, especially its magnitude, is of first importance for therapeutics. In general we must seek to avoid all exertion or excitement ; horses must not be ridden nor worked, and dogs must not hunt, whereby indeed the value of such animals is so much depreciated that they are hardly worth further treatment. Then above all they must have good, albuminous nourishment, that the muscular apparatus of the heart, being compelled to hypertrophy, may have material for formation of new muscle. Of drugs we may give iodide of potassium as a sedative against the hyperkinesis of the heart which arises in the course of its hypertrophy. It is given to horses in doses of  $1\frac{1}{2}$  to 3 drams, and to dogs of 3 to 15 grains.

If, however, the hypertrophy be already complicated with enlargement, and the pulse therefore grown feeble, the exhaustion and weakness of the heart must be combated by cardiac stimulants, as well as by strengthening remedies. Digitalis-leaves are given then in small and frequent doses (horses,  $\frac{1}{2}$  to  $1\frac{1}{2}$  drams ; dogs,  $1\frac{1}{2}$  to 5 grains per diem in infusion). As these tend to reduce the speed of the heart, the pauses be-

tween the various phases of its activity are longer and its muscles gain time to recover and absorb nutriment. Naturally the continuous administration of digitalis requires great caution. Besides digitalis we may mention as admirable cardiacs, caffeine (horses and cattle,  $1\frac{1}{2}$  to 3 drams; small dogs,  $1\frac{1}{2}$  to 8 grains; larger dogs, 8 to 30 grains), and strophantus-tincture (horses and cattle,  $2\frac{1}{2}$  to 6 drams; dogs 10 to 25 drops). Among stimulating remedies, especially if disturbances of circulation appear, we may use wine, camphor, alcohol, ether and veratrine. Good, substantial food acts as a strength-giver. By acting in this way we may at least delay for a time the impending insufficiency of the cardiac muscles, even though a perfect cure of hypertrophy and dilatation only occurs in rare cases, viz., where the cause can be permanently removed, as in idiopathic hypertrophy.

## RUPTURE OF THE HEART.

**Etiology.**—The causes which lead to rupture of the cardiac muscles are, apart from traumatic influences, to be sought in some morbid condition of its walls, which leads either directly, or after the operation of certain other factors, to a solution of continuity of the organ. Among morbid changes of the myocardium must be cited: softening, inflammation of the cardiac muscles, formation of abscesses, weakening of the wall by induration and aneurysms, fatty or senile degeneration of muscular fibres, endocardiac and pericardiac processes, atheromatous degeneration of the aortic walls at their origin, echinococci cysts and worms (spiroptera) in the wall of the heart. The exciting causes are sometimes physical shock, such as a fall, over-excitement during coition, or (in dogs) during operation for ascites, and at other times acute tympanites, as often occurs in cattle, etc.

**Post-mortem.**—Besides the changes mentioned in the myocardium, we find a fissure of varying length in the wall of the heart, very often in that of a ventricle or auricle, and from one to two inches in length. Sometimes either the aorta or pulmonary artery is ruptured at its base. Blood is found collected in varying quantity in the pericardium (haemopericardium).

**Symptoms.**—Rupture of the heart betrays its occurrence either in sudden, apoplectic collapse and death, the animal

often uttering a loud cry, or in staggering, dizziness, trembling, quickened breathing, falling to the ground and convulsions, accompanied by indications of internal haemorrhage. Death in the latter case follows after a few or several hours. No treatment is possible.

PALPITATION OF THE HEART AND SO-CALLED ABDOMINAL  
PULSATION IN HORSES.

**Definition.**—Under this name of "Palpitation" we find quite a series of morbid appearances in horses and dogs described in literature, which are by no means uniform and have met with very different interpretation and explanation. Certainly they cannot all be identified with the neurosis of the heart which in man we call palpitation of heart, but only fall partly under this head. The greater number seem to have nothing whatever to do with primary heart-affection, but to be identical with nervous cramp or spasm of the diaphragm. If we discuss them here all together in the chapter of heart-diseases, it is because they are usually designated palpitation of the heart, and because their pathogenesis is as yet too little known for them to be assigned to a special chapter, and because, moreover, rhythmical, spasmodic pulsation in the region of the thoracic and abdominal cavities is common to them all. Subsequent affections seem to have been described as "Palpitation of the Heart," or "Abdominal Pulsation" in the abundant literature which we possess.

I. **True Nervous Palpitation of Heart**, caused by neurosis of the heart without any demonstrable anatomical changes. The frequent accentuation of the heart-beat which occurs during hypertrophy of the heart, especially with valvular defects, does not come under a correct definition of palpitation, nor the throbbing, laboured heart-beat which accompanies anaemia and leucæmia caused by disturbed nutrition or atrophy of the heart. The causes of nervous palpitation are to be sought first of all in a general nervous temperament of certain animals; while among directly operative factors are great excitement by excessive exertion, severe flogging, hanging by night on the halter, dragging at the head too much in riding, etc.

The *symptoms* of nervous palpitation are: throbbing heart-beat, accompanied by concussion of the costal wall, or even of

the entire thorax, and which may sometimes be heard some paces away; accelerated, small, irregular pulse, quickened respiration, restlessness, fear, trembling, sweating. Auscultation of the heart reveals very loud heart-sounds, which are frequently amalgamated into one. The accentuated heart-beat can also be perceived along the course of the aorta by laying the ear against the animal's back. During the attack the animal often passes faeces, and poses itself for urination. Venous pulse and even visible pulsation of the carotid arteries have also been noticed. In isolated cases, the ailment ended fatally in paralysis of the heart or cerebral apoplexy; but in most cases, recovery takes place after a shorter or longer duration of the attack, although the paroxysms may be frequently repeated. Of considerable importance in differentiating palpitation from kindred affections is an early test as to the correspondence between the throbbing in the thorax and the several heart-beats; the two should exactly synchronise.

**2. Nervous Spasms of the Diaphragm.**—These produce symptoms nearly resembling those of palpitation of the heart, which are often confounded with them by observers, or described as "*abdominal pulsation*." Contractions of the diaphragm may have many causes. C. tarthal and inflammatory conditions of stomach or bowels seem able to produce spasmodic cramp of the diaphragm, either by reflex action, or by transmitting inflammation to that membrane. Thus, marked symptoms of palpitation of the diaphragm were noticed by Brill in conjunction with vomiting; Berghuis, after overfeeding; Oreste, with gastric phenomena, bad quality of food, colic, etc.; Grosswendt, in bowel-inflammation; Leblanc, Wörz and others, immediately after drinking cold water; Boiteau, after eating fungoid hay, in which case they were to some extent an indication of poisoning; and Cartwright, after administration of an aloes-pill. It is also within the range of possibility, that nervous palpitation of the heart may set up morbid contraction of the diaphragm, and that the phrenic nerve, which runs across the base of the heart, may be irritated by abnormal activity of that organ (Haubner-Siedamgrotzky).

The phenomena of spasms of the diaphragm—not inappropriately called also "Chorea of the Diaphragm"—are most peculiar and striking. In the first place we note violent throbs, especially in the region of the left inferior ribs and flanks, which shake the whole body. These are sometimes accompanied

by a subsequent short backward expiration (Boiteau, Hering), and by a dull tone audible some yards off (Simonin and others). These throbs, resembling electric shocks, may be most distinctly felt along the insertion of the diaphragm (Grosswendt), and seem at times to be restricted to certain spots upon the latter (Cartwright). In rare cases they are accompanied by a decided sob (*singultus*). Moreover, they do not synchronise with the heart-beat, but only count from 10 to 15 strokes per minute, seldom more; and in most cases the heart-beat is not at all violent, but normal or even unusually weak. It is evident then that the abdominal pulsations thus described cannot arise from the heart as prime motor. Only after repeated attacks do these abdominal pulsations become synchronous with the heart-beat, but always stronger than they, while the throbs decrease in violence forward, or towards the heart. Here we may perhaps suppose an irritation of the phrenic nerve by the heart movements to occur. Besides these symptoms, a small, frequent pulse is noted, increased and jerky breathing, tremblings, restlessness, yawning, frequent stretching out of a hind-foot with indications of pain. The spasmodyc attacks sometimes only last a few minutes, at others 12 to 24 hours, or even 2 to 4 days, seldom longer (Leblanc observed a case lasting three weeks). *The prognosis* seems to be less favourable than in palpitation. Cartwright noted six fatal cases among 18. The greater frequency of left-hand pulsation (Cartwright records that of 16 cases, 12 were on the left side) may perhaps be owing to the position of the stomach, or to changes therein producing illness.

**Treatment of Palpitation of the Heart and Spasms of the Diaphragm.**—This consists in the administration of anti-spasmodyc remedies, such as morphia (subcutaneous injection of 5 to 8 grains for a horse); bromide of potash and chloral hydrate (6 to 12 drams). In nervous palpitation of the heart, cold irrigation of the region of the heart may be recommended, with irritant inunctions upon the skin, besides derivation to the intestinal canal by the use of purging and even drastic remedies. We must leave it an open question as to whether bleeding, once so regularly adopted, is an operation to be advised.

**NOTE:**—In assuming, as we do above, that the origin of so-called abdominal pulsation is to be sought exclusively in spasm of the diaphragm, we must remark further that in so doing we agree with the

majority of observers. The most eminent of these, such as Apperley and Castley, Besson, Boiteau, Goubeau, Hering and, in later years, Cagny, all maintain this opinion. Confusion only arose when cases of true nervous palpitation of the heart were classed with spasms of the diaphragm. The acceptance of this last-named malady explains easily and naturally the pulsations, sometimes so extraordinarily violent, whereas a mere interruption in the circulation in the posterior aorta (Berghuis, Simonin, Anacker and others) cannot possibly produce this phenomenon, still less so an aneurysm of the aorta. Moreover, a simple contraction of the abdominal muscles (Délaïfond, Cadiot) cannot be considered, as it only produces straining. To deduce these pulsations entirely from the artery of the liver and kidneys is certainly a very naive explanation. It is scarcely necessary to discuss more fully the fact that the palpitation noticed in connection with thrombosis of the iliac arteries and posterior aorta has nothing to do with the malady before us, although they have often been confounded. According to all these data, we must regard the so-called abdominal pulsation of horses as being a neurosis of the diaphragm, nearly related to singultus in man, mostly occurring alone, but sometimes also simultaneously with nervous palpitation of the heart.

**Bradycardia.**—Under this name we understand, in contrast to palpitation, a noticeable retardation of the heart's action, that is to say, of the pulse. Thus in horses, the latter will sink to 14-20, and in dogs to 20 a minute. The causes of this phenomenon are very diverse (valvular defects, dilatation of the heart, enlarged aorta, new growths near the centre of the vagus, great loss of blood, etc.).

## ANEURYSMS.

**Etiology.**—The causes of aneurysms of the aorta, i.e., of circumscribed enlargement of that vessel, are mostly to be sought in morbid changes of the walls by chronic endarteritis and mesarteritis, accompanied by fatty and calcareous degeneration. Such vascular troubles occur in horses most frequently in the mesenteric arteries as a result of the presence of *Strongylus armatus*, being known as worm-aneurysms. (See the par. on Embolic Colic in Chap. III., Vol. II.). Spiroptera sanguinolenta may also produce the same result, as also may traumatic, mechanical influences, such as violent muscular exertion, a blow or a fall, particularly if some arterial disease be already present. A diathesis seems sometimes to be also present for diffuse formation of aneurysms (Raymond).

**Post-mortem Lesions.**—Aortic aneurysms are either sac-shaped, spindle-shaped or cylindrical. In true aneurysms the sac is bounded by the walls of the vessel, and in false ones by those of neighbouring organs, such as the bowels, liver

etc. The size may be quite considerable, exceeding that of the human head; and at times they grow fast to the adjacent organs, e.g., to the stomach, colon, caecum or liver. Aneurysms are commonest upon the anterior mesenteric artery, also at the bifurcation of the aorta, on the abdominal artery, the thoracic aorta, at the root of the aorta, on the coronary and femoral arteries, on the internal maxillary, etc. In most cases all three coats of the aorta share in the dilatation; being thereby atheromatously and fattily degenerated, alternately thickened and atrophied, and showing lamellar deposits of lime. The contents consist of coagulae of fibrin, which are frequently stratified, organised, or already purulently decayed. According to Röll, these may in domestic animals spontaneously heal by shrivelling and cicatrisation. Since the distended aorta naturally presses upon the nearest organs, it compromises the lungs, stomach, viscera, liver, kidneys, and even affects the vertebral column. Finally, it may rupture and empty its contents into the thoracic and abdominal cavities, even into the stomach (Vogel, Bleich) and rectum (Labat and Cadéac).

**Symptoms.**—In most cases aneurysms of the aorta produce no visible signs of disease, or they end suddenly by haemorrhage without any warning symptoms having been noted. The consequences of aneurysms of the anterior mesenteric artery have been already described in the section on embolic colic. Death by internal haemorrhage occurs especially after severe exertion, the animal tottering, displaying great difficulty of breathing, collapsing and speedily expiring. In other cases, peculiar paroxysmal phenomena are noted, without any immediate fatal ending. Thus Lustig observed staggering of the hind limbs in a horse after severe exertion, sudden collapse, extreme dyspnoea, as well as epileptiform spasms, during which the head and neck were bent backwards and the feet stretched out. But in 5 to 15 minutes the animal had recovered. In another case of aneurysm of the pulmonary artery in a horse, Lustig noticed fits of dizziness. Barrier records, in a dog with aneurysm of the posterior aorta, growing emaciation along with great appetite, increasing weakness and extremely hard breathing after the least movement and finally, paralysis of the hinder parts (signs of weak heart). In a horse which had an aneurysm of the thoracic aorta as large as a man's head, Schmidt noticed a humming tone over the vertebral column which synchronised with the

action of the heart, inability to pick up food from the ground, combined with a stiff and constrained gait in the hinder limbs.

**Therapeutics.**—In man, the treatment consists in the application of galvano puncture, which is even extended to the thoracic aorta; in injections of ergotin, as well as internal administration of sugar of lead, or iodide of potassium. These last remedies may also be applied in the case of animals.

#### RUPTURE OF THE LARGER VESSELS OF THE THORACIC AND ABDOMINAL CAVITIES.

**Etiology.**—Ruptures of the larger vessels in the cavities of the thorax or abdomen generally pre-suppose some change in the walls of such vessels caused by chronic endarteritic processes (arterio-sclerotic, or atheromatous changes), or aneurysm. The indirect causes of such rupture are partly physical exertion, such as the drawing of heavy loads, and partly a fall, the act of vomiting, the operation of veratrin medicinally administered, etc. Ulcerative processes in the locality of the artery may also destroy its walls. In rarer cases vascular rupture is produced by the presence of worms in the vessels; thus by strongylus armatus in horses, or spiroptera sanguinolenta in dogs. Finally, animals with hypertrophy of the heart are pre-disposed to rupture their blood-vessels.

**Anatomical Notes.**—Rupture of the larger vessels occurs in the aorta and its branches, in the portal vein, in the posterior vena cava, in the anterior vena cava, and in the pulmonary artery. Most aorta-ruptures affect the root of the vessel near its crescent-shaped valve, when the blood gushes into the pericardium, which, on dissection, is found tightly packed with blood. If only the intima and media be ruptured, the blood flows in between the media and adventitia, forming a so-called dissecting aneurysm.

**Symptoms.**—The signs of rupture of a larger vessel are those of internal haemorrhage, shown by sudden paleness of all visible mucous membranes, thready or imperceptible pulse, weakness, staggering, collapse, with cold extremities. In one case Straub saw epileptiform fits precede the rupture of an anterior vena cava. Hartmann found violent haemoptysis in the bursting through of an aneurysm of the pulmonary

artery into a larger bronchus. Any treatment of such internal vascular ruptures is out of the question.

#### THROMBOSIS OF THE FEMORAL, PELVIC AND AXILLARY ARTERIES.

**Etiology.**—Thrombosis of the femoral and axillary arteries really belongs to the realm of surgery, as its chief symptom is lameness. But as its causes are entirely internal, and it is only a question of larger vessels, it may also be regarded as an internal ailment. Its origin lies, on the one hand, in an affection of the inner membrane of the artery (endarteritis), with deposition of fibrin-coagulae upon the roughened surface and stenosis of the lumen; on the other hand, in embolic stoppage of this branch of the aorta by thrombi which have been floated thither, and have their source in the heart or in an aneurysm.

**Anatomy.**—The aorta is most frequently found blocked with thrombi at its division into the two iliac or femoral arteries, and in the two pelvic arteries, less frequently in the axillary or brachial artery, and only in very rare cases in the lumbar artery. The end of the aorta and the root of the bifurcating arterial branch are mostly widened, the walls thickened, and atheromatosely degenerated, the intima dull, fattily-degenerated, calcareous and covered with ulcers. Within is fixed a thrombus, which is usually stratified, organised and hard, of pale colour, and which more or less narrows the lumen, often leaving only a very small passage. The thrombus often grows forward into the aorta on one side, and into the arterial branch on the other, or adheres to the place of bifurcation. Sometimes the iliac or pelvic artery of one side is entirely filled by the thrombus. Along with this thrombosis a compensatory hypertrophy of the heart is not unfrequently developed as a secondary symptom. Also upon softening of the thrombus, emboli occur in the periphery of arteries in the extremities.

**Symptoms.**—The thrombi just described usually cause no signs of illness during rest. Not until after prolonged running in harness or under the saddle does the peculiar and characteristic aspect of the disease appear.

1. *In Thrombosis of the Femoral Artery*, which mostly affects horses, but cattle more rarely, we notice first of all a gradually

increasing weakness of one or both hind-legs. The animal shows a characteristic lameness, which comes on at intervals. It gives way mostly on one side, has an unsteady gait, strikes the affected foot against the one on the other side, only puts the toe to the ground, or drags the foot behind, trembles, breaks down, and then lies for some minutes on the ground, first sprawling with its legs, and then quite exhausted. At the same time breathing is extraordinarily accelerated, the heart-beat becomes rapid and jerky, the visible mucous membranes much injected, and the whole body is covered with perspiration. The temperature of the affected limb is usually very low, and pulsation is often, if not always, absent from the tibial and metatarsal arteries. After a few minutes the horse rises, and its lameness by degrees passes away altogether. Of importance for diagnosis is the fact that an attack can be experimentally produced by forced movement. On examining the location of the thrombus through the rectum, we find the aorta and its branching artery widened, thickened, unyielding, filled with a hard, elongated body, and generally without pulsation on the affected side. In rare cases the thrombosis is complicated with peripheral venous embolism with gangrene of the limbs.

2. The less common *Thrombosis of the Axillary Artery* reveals itself in simple lameness upon the side affected. During movement the animal begins to stumble, makes false steps, straddles, continually knocks its toe, cannot lift its feet, but quite drags them along, trembles upon the affected side and finally rolls to the ground. We have, however, not noticed any disturbance of breathing, excitement of heart, nor signs of congestion. The animal soon recovers its powers for the time being.

3. *Thrombosis of the Pelvic Arteries* results in paralysis of the rectum, urinary bladder, tail and croup.

**Therapeutics.**—Treatment of the thromboses as above described generally has but little effect, certainly internal treatment (iodide of potassium, alkalis), is almost useless. More effectual appears to be the massage of the thrombus by way of the rectum recommended by Colin and Bayer, viz., careful stroking and kneading of the plug, thus softening it and aiding its resorption; but there is a danger here, that some parts of the thrombus may be embolically carried away. Finally, by methodically exercising the animal, we may

strengthen the cardiac muscles and increase the blood-pressure, and thus secure an improvement of its general condition. Absolute rest for several weeks has in many cases been known to produce a very favourable result (resorption, or organization of the thrombus). On the other hand, in some cases we have seen the thrombus very much enlarged after such a lengthy rest, so that no opinion of real practical worth can be offered on this point.

**Formation of Thrombi in the Heart.**—This arises from disease of the endocardium, viz., in ulcerative valvular endocarditis, whereby weakness of the heart, with simultaneous stagnation of the blood-column, exercises a pre-disposing influence. The thrombi, which at first are small, only give occasion for general disturbance when they lead to the narrowing of an orifice. Symptoms of stenosis of the respective orifices then develop, with subsequent dilatation of the heart; emboli in the lungs and towards the periphery of the body also occur. These heart-thrombi, which usually adhere firmly to the endocardium, must not be mistaken for blood-coagulae in the heart.

#### WORMS IN THE BLOOD. HÆMATOZOA.

**Occurrence.**—Several varieties of worms are found in the blood of various animals, especially dogs, viz.: 1. *Filaria immitis*; 2. *hæmatozoon subulatum*, or *strongylus subulatus*, or *strongylus vasorum*; and 3. *strongylus inflexus* (found by Hering in seals and dolphins).

The most important of these is certainly *filaria immitis*, a round worm, said to have been discovered by Leydy, 1858, in Philadelphia, and identical with the *filaria papillosa hæmatica* of Delafond and Gruby. The fully developed filariae attain a length of from 6 to  $13\frac{1}{2}$  inches and a thickness of 1 mm. They are to be found knotted together to the number of 50 to 75 in the ventricles, in the pulmonary artery, etc. Their embryos live in hundreds of thousands in the blood of dogs, and are on an average  $\frac{1}{2}$  mm. long, by 5 micro-millimètres thick. Lewis found these filariae embedded as knots in the walls of the aorta of an Indian dog. According to Janson, they are dangerous, owing to their wanderings throughout the body (thrombosis of the posterior and anterior vena cava, veins of the liver and kidneys, and of the lumbar, axillary and jugular veins); also owing to their closing the valves between the ventricle and auricle, i.e., by twisting themselves around the chordæ tendineæ.

**Symptoms.**—No matter in what numbers the filariae be present in the blood, they usually produce no signs of disease for a long time. But in some cases they cause apoplectic death by the creation of thrombi in the heart, by stoppage of passages and even rupture of the heart. Emboli of their embryos in the capillaries of the brain appear also to produce apoplexy of that organ. Furthermore, some have observed nephritis, cough, vomiting, pain, epileptiform convulsions, dyspnoea, emaciation, great feebleness, painful lameness of a hind-leg (symptoms resembling rheumatism), signs of lameness in the hind-quarters, haemorrhage in the lungs and bowels (*gastro-enteritis haemorrhagica*), and finally even appearances of rabies.

The course of filaria-sickness is chronic. One may differentiate between a *latent* stage, lasting for months and years without any visible signs of illness, and an *evident* stage (acute or peracute) with the symptoms described above.

**Diagnosis.**—The recognition of filaria-sickness during life is difficult, owing to the general character and multiplicity of the symptoms and their absence in the first stage. Diagnosis is nevertheless much assisted by the observance of two factors. First of all, the complaint, as a rule, only occurs in dogs which have lived formerly abroad (China, Japan). One finds then, on careful examination of the blood, the characteristic embryos of filaria,  $\frac{1}{4}$  mm. long by  $\frac{1}{100}$  mm. thick. This latter examination ought therefore to be made with all imported dogs which show signs of emaciation, paralysis, etc. In one case which we observed, a dog brought up in Berlin was affected, but this was exceptional, and on dissection, no sexually ripe animals could be found, but only embryos.

**Therapeutics.**—Until the method of immigration of the filariae is determined, no treatment is of any use, and even then it can only be of prophylactic nature. The suggestion that they are transmitted by inheritance by breeding from affected dogs is very doubtful. Arsenic is said to have been given in Japan with good results to destroy the parasites in the blood.

**History of Development.**—According to Manson, the mosquito of the tropics in the New World, by absorbing human blood, becomes the intermediate host of the *filaria sanguinis hominis*. As soon as the filaria has penetrated from the mosquito's stomach through its body cavities to the thorax, its movements cease, and it

begins to grow, passing through six stages of development. After the female mosquito has laid its eggs and perished in the water, the filariae break through her skin, and play freely about in the water, so that men may be infected in two distinct ways, viz., by drinking such water, or by being bitten by mosquitoes which have previously sucked blood containing *filaria sanguinis hominis*. In a similar manner we must imagine dogs to be infected. In Japan, where 50 per cent. of all dogs, especially those imported, yearly fall victims to the *filaria immitis* (Janson found it in 86 dissections out of 193), the opinion is general that the embryos discharged with the urine reach the rice-fields, where they collect in puddles, and are again absorbed by the dogs.

*Hustrongylus Gigas* was in one case found in the cardiac cavity of a pig (Fabretti).

## CHAPTER X.

### SKIN DISEASES.

**Introductory Remarks.**—Skin diseases occur in many varied forms in all our domestic animals, especially in dogs, horses and sheep, but also in cattle, pigs, goats and cats, and are also very common in poultry. This multiplicity of form in skin troubles has for ages led to the establishment of a great number of separate diseases (some of them falsely taken from human medicine), and to many different names, not all of them well chosen. Names such as ring-worm, dry-rot, erysipelas, St. Anthony's fire, scabies, mange, greasy heels, scall, itch, scurf, which were often applied indiscriminately to the same complaint, have survived to our own day, and have caused not a little confusion of ideas. Although it would be better to drop all these old names entirely, yet in treating of skin diseases one cannot, for the sake of clearness, altogether dispense with their former names. It is, however, imperative to discard some of them, and to try to group the great number of separate skin diseases under certain general heads. Thus we propose to treat several forms, which have hitherto been separated, from the stand-point of eczema, following herein the example set by Hebra in human medicine, who has very much widened the conception of eczema by including therein some apparently heterogeneous diseases. Furthermore, the expression ring-worm, or dry scab, we shall only apply to vegetable parasitic skin affections, as proposed by Gerlach, and the term "mange" only to skin eruptions caused by mites or the acari of scabies.

A strictly scientific sub-division of skin diseases is much more difficult with domestic animals than with man, owing to these

skins being covered with hair and pigment. For instance, diffuse or localised reddening of the skin can only be considered in animals whose skin is free from pigment, or in portions of their skin which are pigment free. No system of diagnosis can be built up on the basis of pathological changes because the same lesions may occur in the most diverse skin diseases, and, vice versa, the same skin disease may in its course reveal the most varied pathological aspects. Thus, the term "scabby nodules" indicates no clinical unity, as nodules occur in quite a series of skin complaints. While, on the other hand, eczema exhibits several lesions (hyperæmia, nodules, blisters, vesicles, pustules, scabs and incrustation). It also does not seem opportune to divide skin diseases into acute and chronic, for many acute conditions of the skin, such as eczema, for instance, may often become chronic. A further division into idiopathic forms, or such as are caused by external injuries, and symptomatic, arising from internal morbid causes such as infectious disease, is also not unobjectionable, for some complaints, for instance urticaria (nettle-rash), a clinically sharply defined skin affection, may be produced externally (by insects) and internally (by strangles, swine erysipelas, nettle-fever, and pleuro-pneumonia contagiosa or murrain). Etiology is therefore by far the most important principle for subdivision in skin disease, but even this cannot always be adhered to, as in many skin affections their etiology is as yet unknown.

If we therefore adopt as most suited to our purposes the division into parasitic and non-parasitic skin diseases, we would first point out that the province of vegetable-parasitic skin eruptions is as yet very imperfectly fixed, and that the opinions of authors differ greatly as to the part which the various fungi play in the production of the several diseases, especially as vegetable micro-organisms may chance accidentally upon an already diseased skin.

After these preliminary remarks, we propose in the following chapter to adhere in the main to our subdivision into parasitic and non-parasitic skin diseases, the former of which we further divide into vegetable and animal parasites. The greater part of the symptomatic eruptions (variolæ, foot-and-mouth disease, etc.) arising from internal causes, as distinct from idiopathic affections, which we may also term "Exanthemata," and which "blossom-out," so to speak, from some internal disease, have been already treated among infective diseases in our first

volume. Finally, a good many skin affections such as erysipelas, phlegmon or cellulitis, panaritium, new growths upon the skin, etc., must be left to surgery.

#### I.—NON-PARASITIC SKIN DISEASES.

##### *Erythema of the Skin. Dermatitis Erythematosa.*

**Definition.**—The simplest form of skin affection consists in hyperæmia of the papillary bodies and superficial papillæ of the skin, which is known as erythema of the skin and occurs sometimes diffusely, and at others in spots and locally (roseola). Such a condition is, however, only visible in domestic animals upon the unpigmented portions of their skin. We find it therefore most frequently in sheep, pigs, dogs, white-born horses, piebalds, as well as on the white parts of horses and cattle, about the head and extremities. The only perceptible symptom of erythema is a more or less localised or diffuse redness of the skin which, in contrast to haemorrhagia (petechia, ecchymosis, purpura) can be made to disappear for a time by pressure with the finger. This redness is sometimes accompanied by itching (pruritis). If long continued, the erythema produces a more or less considerable desquamation of epidermis. In contrast to eczema there is no weeping.

**Etiology.**—Erythema of the skin occurs either independently or forms the introductory stage of other skin diseases. In the former case it generally soon disappears, leaving no pathological change behind in the skin. Its causes are :-

1. *Mechanical influences* upon the skin, such as pressure, friction, shearing in sheep, horses and dogs, whipping swine in transport, etc., so-called *erythema traumaticum*.

2. *Chemical irritants*, slight erosions, inunction of carbolic, tar, cantharides or tartar emetic ointments, or of black soap, sinapisms, etc.; decomposition of urine on the skin near the place of discharge during paralysis of the hind legs; insects' stings; the effect of fungi upon the skin. This is known as *erythema toxicum*.

3. *Thermal influences*, excessively high or low temperature (first stage in scalding, burning or freezing); the effect of direct sunshine upon the non-pigmented skin (so-called sun-scald of horses). Known as *erythema caloricum* and *solare*.

**Therapeutics.**—Treatment is only occasionally necessary.

Beyond removing the causes, one may apply as a relief for intense itching, fomentations of lead-water, or sprinkle a powder of starch and oxide of lead, or spread a lead or zinc ointment; or finally, if the pruritis be very intense, apply a 6 per cent. solution of nitrate of silver.

**Eruption produced by Buck-wheat** (*Fagopyrismus*).—This is a peculiar form of erythema of the skin, although lying somewhat outside its simpler forms. It may run its course as true erythema, but just as frequently represents a complication of erythema with erythematous inflammation of the skin (*i.e.*, inflammatory infiltration of the cutis or inflammatory oedema of the skin), or with vesicular, bullous, phlegmonous, erysipelatous or even gangrenous dermatitis. In general no strict line can be drawn between erythema of the skin and an erythematous inflammation. The original cause is to be sought in the eating of buck-wheat (*polygonum fagopyrum*) and of other *polygonum* species (*polygonum persicaria*), and not only of the green buck-wheat, and especially at the time of flowering, but also of the grains, straw, stubble and chaff. Sunshine seems also to be necessary as a second factor in the matter. We can therefore best imagine the disease originating as follows: the parasitic fungi living on the buck-wheat, or their irritant products of metabolism, come in contact with the animal's skin, but can only affect them upon their non-pigmented portions, and then only when they are first made sensitive by the sun's action in producing erythema solare. Whether insects can produce this buck-wheat eruption in some instances (such fields are much favoured by bees), or whether some animals are peculiarly sensitive to the influence of the buck-wheat is uncertain. The sickness is commonest in white and white-spotted sheep, especially lambs, also in pigs; is rarer in goats and cattle, and very rare in horses which have unpigmented portions of skin. The more buck-wheat an animal has eaten, and the more it has been exposed to the sun's heat, so much the more violent is the outbreak. Black animals or such as are blackened over, do not contract it. The same, too, with those kept in the stable, or only pastured in cool weather, or which feed in the shade. On the other hand the eruption appears even if they have discontinued eating buck-wheat for eight to ten days, or even three to four weeks, and are then taken out into the blazing sunshine. On returning to the stable, the ailment soon vanishes, but returns on coming back to sunlight. In winter the eruption seems to be restricted to mere itching and burning.

The phenomena consist in intense redness (erythema) and inflammatory swelling (dermatitis erythematosa) of the head, especially the ears, face and eyelids, which may reach the inter-maxillary space and throat, and are accompanied by intolerable itching. The animals naturally grow irritated, shake their heads, rub themselves, run about restlessly, and even manifest extreme signs of inflammation of the brain, the same as in mania. Sometimes little blisters arise on the red and swollen skin the size of a lentil, or from that to a pea, which on bursting discharge a yellow fluid and then heal, leaving a scab. This is the so-called head or pock erysipelas of sheep. At the same time, one may observe in graver cases some disturbance of breathing (erysipelatous inflammation of the mucous

membrane of the air passages), general feverishness, digestive trouble, as well as symptoms of brain affection : stupor, heaviness, rotary movements, spasms, great excitement, etc., probably during the spread of the erysipelatous processes from the skin of the head to the meninges ; in such cases the ailment may even result in death. The epileptiform fits observed by Rabe and others in horses and pigs, without any accompanying skin affection, complicated with signs of vertigo, seem to indicate the presence in buck-wheat of some narcotic poison.

*Treatment* consists, as regards prophylaxis, in only taking white sheep to graze on buck-wheat pastures on cloudy days or towards evening, or else serving the buck-wheat exclusively when under cover. When once the disease has showed itself, the animals must be driven without delay to a shady place or, better still, back to their stable. Local treatment is only necessary in severer complications, especially in erysipelatous inflammation of the skin of the head with cerebral and general symptoms. One should apply cooling bandages, wash the head with creolin water, olive-oil in combination with lime-water as well as anti-phlogistic remedies, such as tartar emetic, neutral salts, etc.

## ECZEMA.

**Definition.**—Eczema may be best defined as a dermatitis, manifesting itself in various stages of development and intensity. In its nature and course, therefore, eczema corresponds entirely with inflammation of other organs, such as the mucous membranes, whence it was formerly regarded as "catarrh" of the skin. Just as there are various forms of one and the same catarrh of the mucous membranes, corresponding with the degree of inflammation, viz., serous, mucous and purulent, so, too, the clinical aspect of eczema is quite varied according to the acuteness of the inflammation and the nature of the affected portions of skin. These multiform outward appearances of eczema led formerly to the classification of a great number of apparently different and independent skin diseases under this name. This can be the more easily explained, seeing that the selfsame skin affection presents seemingly great differences according to the kind of animal or the locality, and that the simplest of such affections might be vastly changed in appearance by the animal rubbing, scratching or gnawing. Also on the further ground that identical causes might produce quite different aspects of disease according as the individuals varied in sensitiveness, or according to place affected, or the constitution, age, etc., of the animal. But the fixing of so many separate forms of skin disease has been productive of much confusion and obscurity, that a reduction in their number is

urgently needed, both for purposes of instruction and for consideration of the diseases from a common stand-point.

**Stages.**—Like every other inflammation, eczema may pass through various stages. It will be best to follow the rule adopted in human medicine and adopt with domestic animals the following six stages of simple eczematous inflammation of the skin, according to the nature and composition of the matter discharged.

1. *The erythematous stage*, which consists in hyperæmia of the skin with superficial formation of exudation, in consequence of which the epidermis is more abundantly formed and later on becomes exfoliated in the form of scales or little bran-like plates. In the chronic course of erythematous eczema it is called squamous eczema, owing to the excessive desquamation of the epidermis. Very often therefore this squamous form occurs as the final stage of the entire process of eczema. In this class we include the skin diseases of our domestic animals known under the names of "bran" or "scaly tetter," also "hunger-mange," "psoriasis" and "pityriasis"; and some stages of mallenders and greasy-heels of horses.

2. *The papular stage.* The small red nodules characteristic of this stage show under the microscope that they have been produced by small-cellular infiltration and serous saturation of individual papillæ, with swelling of the same. This papular eczema corresponds to the "lichen," "rash" and "sun-scabies," and to some cases described as "nodulous," "pimply" or "heat" eruptions.

3. *The vesicular stage* develops directly from either one of the foregoing, when the serous exudation in the nodules is so abundant as to burst the yielding cells of the rete Malpighi, so that the fluid contents can penetrate almost to the surface of the skin, immediately beneath the stratum corneum. This vesicular eczema represents to some extent the type of eczema, and is therefore commonly known as eczema simplex, and corresponds to the so-called "herpes" of our domestic animals. The so-called "heat-rash" of horses belongs probably also to this stage.

4. *The moist stage*, or weeping stage, which sets in after spontaneous or artificial (scratching) opening of the blisters, and represents the eczema rubrum, the "weeping" or salt scab. Eczema rubrum occurs in dogs most frequently as the rash of surfeit.

5. *The pustular stage*, which develops directly, or in consequence of the transformation into pustules of the vesicles filled with serum. In such cases the skin is often changed over a considerable area into a suppurating surface when the pustules have burst. This is the so-called eczema impetiginosum, and corresponds to "pustulous rash," "scall" or "scurfy eczema," which is common in dogs and horses.

6. *The scaly stage*, which is a drying-up of the exuded matter, either in the vesicles or pustules, or upon the weeping, scabby surface of the skin. This scaly eczema corresponds to "squamous" or "scurfy rash" in our domestic animals. This stage is followed in some cases, especially such as are chronic, and as a final stage, by the eczema squamosum mentioned under No. I.

The above descriptions explain clearly the numerous varieties of eczema. But it is by no means necessary that the disease pass through all these stages. An early stage may pass directly into the last, or the eczema may long remain persistently in the same stage. But we must not omit to mention that, owing to the pigmented or hairy skins of the animals, it is easy to overlook some of the rapid stages, such as the papular and vesicular.

The various eczemas of individual domestic animals are as follows :

#### A.—THE VARIOUS KINDS OF ECZEMA IN DOGS.

**Occurrence.**—Eczema is the commonest of all skin affections, and indeed one of the most frequent ailments of the dog. According to our experience at the Berlin dog-hospital, one-twelfth of all dogs suffer from it. But it is mostly restricted to certain parts of the body and occurs oftenest along the back, beginning at the root of the tail or on the neck, and spreading thence both lengthwise and sideways. Other favourite spots are the outer surfaces of the extremities and the rump. It appears much more rarely on the lower breast and belly, on the knee-folds or elbow-joints or upon the protuberances of the astragalus. It is most rare upon the head. Old dogs which live much in the house sicken most freely, as well as young, tender-skinned, pampered and therefore fat ones. As regards race, pugs, fox-terriers, Danish dogs, mastiffs and the Leonberg breed seem to be most susceptible.

**Causes.**—Eczema is usually set up as the direct result

of some external irritant acting upon the skin, such as dust, mud, fleas, lice and insects, or of repeated friction and long-continued pressure on certain portions of skin, etc. The favourite place for eczema, the back, is specially exposed to dirt, dust and fleas. Washing or rubbing with soft soap (green or black) often causes eczema in dogs. The fact that an external cause can in many cases not be assigned has led to the presumption of an internal origin. In this way injurious food, digestive disorders, polyæmia and general bodily debility have all been blamed. But these factors can only be regarded as pre-disposing causes, and a direct local irritation must always be presumed.

**Symptoms.**—The symptoms of acute eczema in dogs are extraordinarily multiform, and a description suited to all cases is quite impossible. The appearances are quite different too, according to the stage of the disease.

1. In the initial stage, which, it is true, usually passes unobserved, we see bright red spots, the size of a pin's head, upon the non-pigmented parts of the skin, which quickly grow darker, forming a nodule or pimple in the centre of each, while the hyperæmic back-ground increases, the spots gradually run together, so that the affected skin becomes spongy, turgescent, and of a higher temperature. The hair around these pimples stands on end in tufts; a slight touch of the skin sets up itching, and a harder touch gives pain. As the pimples soon disappear we often only perceive the aspect of erythema or of erythematous inflammation. In this stage the eczema may heal, or may remain some time stationary, though this is rarer. But its condition is often much changed by rubbing and gnawing on the part of the dog, so that the pustular and scaly may follow immediately on the papular stage.

2. But if the eczema run its regular course, small blisters develop from the pimples. These are sometimes isolated, at others grouped, and are at first full of clear fluid. As they are seldom larger than a hemp-seed and very readily burst on being scratched, they are difficult to find on a dog's hairy skin and are frequently not observed. They partly dry up and are covered then by a slight scab, but some burst or are scratched open and then form minute foci of inflammation, above which the hair becomes loosened and falls off. If the eczema heal when in this stage, the fluid in the small vesicles dries up into small crusts, beneath which the inflammation recedes and the

skin is restored. Upon the darker parts of the skin the position of these blisters is shown for some time after by the patch being poorer in pigment and devoid of hair.

3. But the eczema is not frequently inclined to spread superficially, and by confluence of many blisters we obtain spaces without both outer skin and hair, and which are very much reddened and wet (*eczema rubrum*). These are covered with serous, sero-fibrinous and even purulent exudation, are exceedingly sensitive to touch and readily attack the adjoining sound parts (*corrosive rash, eczema exedens*). Or, in consequence of the animal's rubbing, the eczema progresses to an intense purulent and haemorrhagic dermatitis. The cure of *eczema rubrum* occurs by drying-up of the exudation (scaly stage) and regeneration of the subjacent epidermis from its intact peripheral edges.

4. Not unfrequently the vesicles change into pustules, and we then have a pustular eczema. The blisters enlarge and their contents grow dull and become purulent. Like the blisters, the pustules also stand isolated or are spread in groups, and are also not easy to find on the hairy skin. But they often run together, producing purulent patches of various size. The hairs then bristle, stick to each other and are glued with matter into stiff tufts, become felted, are easily pulled out or fall out of themselves. The skin thus laid bare is covered with a yellowish to greenish-yellow, sticky, creamy pus, is very painful and readily bleeds when touched, the skin itself becoming thicker (*eczema impetiginosum*). Healing follows by drying-up of the purulent exudation into scabs and crusts, beneath which suppuration may continue for some time. In rarer cases the process goes deeper and develops to a phlegmonous inflammation, as well as to ulcerous destruction of the cutis, the latter especially when the parts have previously been much rubbed or gnawed. Hairless places then remain behind, the skin being indurated and thickened, and covered with abundant scales. Quite similar localised purulent inflammations of the skin occur, without any previous eczematous blisters or pustules, as a direct result of strong mechanical, chernical or thermal irritation.

5. Eczema may in all stages become chronic. The most important factor here is to prevent the animals from rubbing the places affected. Generally the changes left behind after severe eczema cause a predisposition to subsequent acute outbreaks, which are commonest in the summer months, because the afflux of blood to the skin is then greatest. The changes

produced in the skin by chronic eczema consist in persistent hyperæmia, higher temperature and thickening. Thus the skin of the back on affected portions often feels to be three or four times its usual thickness. The surface, too, of the thickened parts seems smoother and more shiny, and often a permanent and very considerable proliferation of epidermal cells takes place, which then fall off as white to lead-grey scales (*eczema squamosum*). Gradually the skin becomes harder and dryer, rigid; if laid by hand in folds, it remains so for a long time; the hairs bristle in all directions, are only thin and look dry and pinched, or are even lacking entirely or curled like wool (compression and atrophy of the hair-papillæ and their vessels by new growth and retraction of the connective tissue). Such patches of skin often wear a granulated aspect, whereby the hypertrophic papillæ of the skin protrude in the form of thick papillary proliferations above the outer circle.

The general condition of animals during eczema is as a rule unaltered beyond continuous excitement and unrest and a frequently manifested and extreme thirst. In cases of long duration, however, or with repeated relapse, gradual emaciation results, caused by the long-continued irritation, the increased waste of fluid and bodily heat, as well as the persistent reflex excitement transmitted from the skin to the entire nervous system. In feeble or very young animals this may develop to cachexia and death. Acute eczema may last from one to three weeks; the chronic form for months and indeed, with shorter or longer remissions, even for years, and is frequently incurable.

**Differential Diagnosis.**—Eczema may be mistaken first for a purulent inflammation of the skin caused by a wound, excoriation, erosion, scald or frost-bite; also for true herpes, especially herpes tonsurans, and finally for an acarus eruption or mange produced by mites.

1. *Traumatic dermatitis* exhibiting localised, reactive processes which are accompanied by suppuration or detachment of ichorous scurf or scabs of dry gangrene, etc., beginning with gangrenous destruction of a portion of skin, and secondary to which a suppurative, granulating dermatitis occurs. The papules, vesicles and pustules characteristic of eczema are entirely absent. But since these latter are occasionally lacking also in eczema, a differentiation may now and then be difficult.

Very frequently eczema changes into traumatic, suppurative dermatitis as a result of persistent rubbing by the animal.

2. *True ringworm (tinea tonsurans)* may generally be distinguished by the rounded shape of the eruption, by frequent absence of all papules, vesicles and pustules, and usually, too, of itching, by its quality of being transmitted and finally by microscopical examination.

3. *Follicular mange* occurs mostly on the head and legs, is infective, has its own typical morbid aspect, and is much more difficult to heal than eczema, or indeed incurable.

4. *Sarcoptic mange* occurs chiefly on the soft-skinned and less hairy parts of the hypogastrium, on the lower chest, the inner surface of the thighs, about the elbows and at the base of the ears; it is contagious and usually accompanied by very intense itching. But we must admit that the clinical aspect does not always suffice for diagnosis, and that both follicular and sarcoptic mange may much resemble a non-parasitic eczema. In such cases only the microscope will enable a positive diagnosis to be made.

**Therapeutics.**—The treatment of eczema varies both according to stage and individuality. The first thing to do is to remove the causes. It is certain that in most cases it would heal of itself if the abnormal irritation caused by the itching could be corrected. It is here that with most animals the chief difficulty arises. In many dogs this pruritis is so intolerable, and the attempts of the animals to rub themselves are so ingenious, that it is found impossible to secure this first condition of speedy recovery, in spite of muzzles, bandages, collars, boots, etc.

1. In the *lighter cases and earlier stages* of eczema (erythematous, papular and vesicular) antiphlogistic, emollient, anodyne, and protective dressings should be applied. We use either a simple zinc-salve or a freshly made lead ointment, especially Hebra's salve in its newer form: equal parts of litharge and paraffin ointment. As a protective powder for dusting on which will remain a long time lying between the hairs, especially on the ridge of the back, we recommend a mixture of zinc-oxide and starch (1 to 4). The otherwise excellent white precipitate ointment (*unguent. hydrargyr. album*) must only be very cautiously used on dogs, owing to the risk of their licking it off.

2. In *excessively weeping or impetiginous* eczema we have  
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long used with great success a 6 per cent. watery or spirituous solution of nitrate of silver, which, after first washing the weeping or suppurating place, we lay on with a brush, when a slight dry eschar or slough is formed, beneath which the healing process rapidly occurs. Among ointments we use most frequently a nitrate of silver salve (1 : 10-20 paraffin ointment); tannin ointment (1 to 9) and creolin-zinc salve (1 to 9). Besides these, dusting with absorbent desiccant powders acts excellently in persistent weeping. We prescribe, for instance, a mixture of dermatol and starch in the proportion of one to ten; or of creolin with boric acid (2-4 : 100.)

3. For *chronic eczema* our chief remedies are tar and creolin. The former should be used either in solid form or as tar-spirit or in the form of a tar liniment in combination with soap potash, which latter is especially efficacious in chronic impetiginous eczema of the back. It is wise to clip off the hair before application and to protect the places afterwards by a covering of leather or some effective bandage to prevent scratching and licking (carbolic poisoning). Leave the tar on for four to eight days, and then remove the dry scab as tenderly as possible; if needed, repeat the inunction. Tar liniment has the advantage that it can be somewhat easily removed by bathing. Instead of tar we have for many years used creolin, which possesses the healing qualities of tar, with but slight risk of poisoning; viz., a creolin liniment (creolin, potash soap of each 3 oz., spirit 1½ oz.), creolin (1 : 10-20) and creolin ointment (1 : 10-20 paraffin ointment). For eczema squamosum we also use either the tar or the creolin liniment as above, and the tar or creolin spirits (1 : 10-20). Among other remedies we have also used with advantage chrysarobin (chrysarobini 1 to 4 scr.; unguent. paraffin. 5 drams), oxynaphthalin (1 : 10) as well as ichthylol. As an excellent astringent Trasböt recommends iodine-glycerine (tincture of iodine and glycerine 1 : 4).

But these by no means exhaust the list of remedies used and recommended for eczema in dogs. We might mention also naphthol, anthrarobin, resorcin, naphthaline, pyoctanin, dilute nitric acid (1 : 5-10) the oxy-naphthalic acids, etc. But to enumerate them all would lead us too far. Among internal remedies for obstinate chronic eczema we would mention arsenic. We have very often used it and sometimes with good effect. It is usually given in the form of Fowler's solution, 5 to 10 drops at once, for weeks together. At the beginning

of recovery, good may be done during certain stages of the malady by the use of laxatives, as then the drying-up process proceeds more quickly, especially in very weeping eczema. We have never noticed any good result from special dietetic treatment, such as withholding flesh-meat.

#### B.—ECZEMATOUS SKIN DISEASES IN HORSES.

**General Remarks.**—The non-parasitic skin diseases of horses are for the greater part purely eczematous in their nature. If we bear this fact in mind, an understanding of the many forms of dermatitis described as special eruptions in horses will be greatly facilitated. This will certainly render much of the old nomenclature superfluous, a consummation devoutly to be wished as an aid to comprehensive study of these skin diseases. We reckon among eczemata the following skin affections, which have hitherto been regarded more or less as independent forms of eruption :

1. As *papulo-vesicular eczema* are to be regarded : lichen, herpes or tetter and partly also those skin diseases described as summer-mange, saddle-mange, heat-eruption, scaly nodules and pimply or tuberculous eruptions.

2. As *squamous, chronic eczema* we must consider pityriasis and psoriasis.

3. The so-called mane and tail "mange," as well as the disease of the hair known as plica Polonica, are a *chronic and frequently pustular, scaly eczema*.

4. The malady known as greasy heels in the bend of the pastern, as well as that called mallenders and sallenders in the bends of the carpal and tarsal articulations, both represent a *localised eczema*, which at first is *weeping* and later becomes *scaly*.

#### I.—PAPULO-VESICULAR ECZEMA OF HORSES.

(*Herpes, Lichen, Heat-Pimples, Summer-rash, Summer-mange, Saddle-mange.*)

**Etiology.**—The papulo-vesicular form of eczema in horses is usually restricted to certain parts of the body. More rarely the eczema is universal and spread over the whole body, as in the so-called heat-rash in summer (summer-mange). Those parts which sweat most freely are more easily attacked, because more subject to mechanical action, viz., the head (bridle), the

sides of the neck up to the shoulder, the parts on which the collar and harness rest, the back and sides of the breast (saddle, belly-band and traces) and the croup (breeching, tail-strap). Besides these external factors, youthfulness, a fine and tender skin, as well as the season for change of coat, may all be predisposing influences.

**Symptoms.**—The most characteristic feature in papulo-vesicular eczema is the presence of nodules which arise on the spots named in great numbers, irregularly grouped, and at first can only be detected by stroking the hand over the place. In size these nodules vary from a millet-seed to a pea. Their consistency is at first somewhat soft, but they become afterwards harder and firmer. The skin round about them is rather higher in temperature, and may even appear slightly swollen. If laid in a fold and pressed lightly, it is found to be more sensitive. The hair upon these pimples is bristly. During the stage of efflorescence and of healing a moderate degree of itching is present, but is absent during the height of the disease. In the later stages the tops of the nodules are covered with small scabs, owing to a serous exudation beneath the epidermis (*vesicular stage*), whereby the pimples themselves become smaller and harder. These little scabs glue the hair above them into small tufts, which afterwards fall off, or are rubbed off, with the scabs. This leaves small, hairless patches behind, which on the unpigmented skin are slightly reddened, and on the pigmented parts covered with light-grey scales of epidermis. This scaling and desquamation of superficial epidermis may, when exudation is abundant, be very extensive (so-called scaly rash). If the inflammatory process has gone very deep, lighter, thin-skinned bald places (so-called scaly nodules) remain upon the darkly pigmented skin.

**Treatment.**—The eczema just described is very harmless and usually heals of itself after its various stages have been run; the hair also grows afresh by degrees upon the bald patches. Treatment is, therefore, generally not needed. But if such be required, we should endeavour to soften the scabs and scales with glycerine or pure fat, or to stimulate the growth of the hair by rubbing in spirituous remedies. Spirits of tar and of creolin are the best for this purpose (1 : 5—10). Ichthyol is also recommended. In rare cases the treatment is very tedious, or the eczema, particularly when universal, defies all treat-

ment. The causes of this incurable eczema, enduring for months and years, is not known.

**Prurigo.**—Under this name we indicate a papular, chronic and violently itching skin eruption in man, which attacks especially the longer sides of the legs, beginning almost exclusively in early childhood, and being obviously inherited. After lasting for years, it leads finally to characteristic permanent changes of skin (thickening and pigmentation). Prurigo can seldom be cured, and many succumb to the prolonged intense itching, the constant excitement and gradual exhaustion. This disease, which can be defined with the utmost precision in man, is said by almost everybody to occur also in our domestic animals, in whom it is spoken of as "itching-eruption." But in our opinion there are no true grounds for assuming the existence among domestic animals of any complaint corresponding to the prurigo of man. The aspect ostensibly observed in various animals (horse, dog, cattle, sheep) does not agree with prurigo of man either in its causes or its phenomena and course. Indeed, we can find nothing that is characteristic in the descriptions of itch-eruption in animals. We have never seen a case of true prurigo in any animal, and are rather of opinion that a small part of those described as prurigo are nothing else but papular eczema, the descriptions exactly agreeing therewith; and that the major part, especially the chronic and obstinate cases, should be classed as mange. We have, therefore, not included prurigo among the skin diseases (dermatosis) in the interest of greater uniformity of treatment.

**Pruritus.**—This is a complaint in man which consists in itching without any demonstrable anatomical change in the skin, affecting chiefly the longer sides of the extremities, the palms of the hands, soles of the feet, the genitals and region of the anus, and which is evidently of nervous origin. Whether any pruritus not caused by mange ever occurs in our domestic animals is very doubtful, in spite of many opinions to the contrary. But in seven isolated cases of dogs we believe we have observed an ailment very similar.

## 2. CHRONIC SQUAMOUS ECZEMA OF HORSES.

(*Bran and Scale Eruption, Hunger-mange.*)

**Etiology.**—Squamous eczema is treated in some text-books as an independent skin disease under the names of "scale and bran scabies," and identified with psoriasis and pityriasis of man, though this is an error. The most diverse forms of eczema may obviously become squamous during a prolonged course, thus giving the idea that they are peculiar forms of dermatitis. Their causes are, therefore, in general the same as those of eczema. Dirt and lack of attention to the skin, together with bad conditions of nourishment (so-called "hunger-mange"), seem especially to favour the disease. Of the operation of fungi nothing certain is known.

**Symptoms.**—The most important morbid phenomena of squamous eczema consist in an accumulation of small scales, like flour or bran, upon the skin, beneath which the latter suffers chronic change, viz., thickening, discolouration and induration; there is usually only slight itching. Such accumulations occur mostly about the head, especially over the eyebrows, upon the inner surface of the muscles of the ears, then on the neck, base of the mane, shoulders, elbows, haunches, root of the tail and the flexor surface of the carpal and pastern joints. In these latter cases squamous eczema is spoken of also as a form of mallenders and greasy heels. It is a quite harmless skin affection, whose course is chronic, and may last months and years. But it not rarely heals spontaneously.

**Therapeutics.**—Treatment consists in careful attention to the skin, softening the scales with green soap, or application of tar or creolin preparations (salve, liniment, spirits). Tar and creolin seem to change the chronic inflammatory process into an acute one, which rapidly heals. Chrysarobin and ichthyol ointment has a precisely similar action (1 : 10).

**Psoriasis and Pityriasis.**—There is no ground for designating scale and bran scabies by these names with reference to the same diseases in man.

1. *Psoriasis in man* is an infiltration of the papillary body, with excessive development of horny substance on the superincumbent epidermis, whereby at first red nodules (*psoriasis punctata*) with horny, asbestos-like scales (*p. guttata*) appear, which attain later the size of a crown piece (*p. nummularis*), and even coalesce, and may cover considerable areas: These patches often heal first from the middle, leaving ring-like marks, or, if many run together, resembling garlands (*p. annularis et gyrata*). Favourite spots are the longer sides of the elbow and knee-joints, the hairy scalp, the forehead and the ears. We have never seen any skin disease like this in our domestic animals.

*Pityriasis in man* is an extraordinarily rare skin affection, which displays redness and scurfiness of the skin, sometimes over the whole body, and leads to atrophy and even gangrene of the skin, emaciation and death. Schindelka observed an eczema in pigs which he held to be identical with pityriasis rosacea.

## 2. CHRONIC IMPETIGINOUS ECZEMA ON THE LONG-HAIRED PARTS OF THE HORSE.

(*Mane and Tail-Scall or "Mange," Plaga Polonica.*)

**Occurrence.**—We meet with eczema in its various stages, particularly the more advanced ones, such as the weeping

and pustular, very often in horses upon the parts of their bodies covered with long hair, chiefly upon the edge of the nape of the neck (mane or neck-scall), on the tail (tail-scall), and more rarely on the head (head and lip-scall). The hair also participates secondarily in the consequences of this inflammation and disturbance of nutrition, and either sticks together and atrophies (elf-locks, plica Polonica), or else largely falls out (rat-tail).

**Etiology.**—The causes of impetiginous eczema upon the long-haired parts of a horse's body are purely local. First comes the uncleanliness and defective care of the skin, which permit the accumulation of dust upon these parts (that of hay, for instance) and of other irritating matter from without, and which afford especially good quarters for numerous parasites (lice, hair-mites). Eczema is consequently most common in bad and neglected establishments, and in those lands where the proper care of horses is purposely neglected, owing to certain mistaken or superstitious prejudices, as in Poland, Russia, Tartary ("Polish tuft"). On the other hand, excessive attention may lead to eczema, e.g., too frequent washing of mane and tail with soap; when the soap is not always quite washed out again, it may set up the inflammation by repeated and continued irritation. A prolonged state of wetness, after persistent rain or too frequent washing, may set up a maceration of the epidermis beneath the thick hair by the consequent warm moisture, as well as a decomposition of the normal skin-secretions (sebum, sweat), and may thus become a direct cause of eczema, like the rain-rot of sheep. To this the skin in such spots seems to be especially pre-disposed, being fine and tender, because well protected. Finally, previous inflammatory processes in these parts often leave some after-effect behind them, which predisposes to their recurrence (*eczema squamosum*).

**Symptoms.**—The incipient forms of eczema upon the long-haired parts naturally remain concealed, especially when little attention is paid to the mane and tail. Only when the products of inflammation have gathered and decomposed and the complaint is already far advanced, does the owner notice the altered look of the hair and the persistent and violent rubbing and gnawing of the horse. The changes are then mostly as follows:—The skin at the root of the hair shows over a larger or smaller area a coating which is sometimes serous, at others

purulent, or bloody or finally scabby, and which has replaced the epidermis. Occasionally blisters and pustules may be observed. The hairs are stuck together by a sweaty or fatty matter, which is greasy, sticky, dirty and malodorous, often baked together, as it were, into balls and lumps as high as the hand, or felted into tufts and plaits (elf-locks, plica Polonica). In time the nature of the hair becomes changed by the spread of the inflammatory process to its papillæ. It grows thinner, curls more easily, and when in this weakened state, felts and tangles more readily. In places which the animal can easily rub or gnaw, such as the tail, the hair becomes atrophied and falls out; and in course of time sclerosis of the skin sets in upon such spots, with subsequent depression of the hair-papillæ, ending in permanent loss of hair (rat-tail). The skin consequently grows thicker, more brittle and harder, and then passes into the chronic inflammatory stage of scaly eczema.

The duration of the process can in this manner become very tedious, and its cure difficult if not impossible.

**Treatment.**—In addition to thorough cleansing with soap, this consists in drying the weeping and impetiginous places. The better to do this, cut off the hair if possible close to its roots. Then apply to the now accessible skin a liniment either of creolin or tar, or else astringent or desiccant powders (iodoform with powdered oak-bark; or, finally, a six per cent. solution of nitrate of silver. The latter has a tendency to discolour light hair, to which fact attention should be called before using it. It is generally a good while before a complete cure can be secured.

**Elf-locks.**—In the foregoing we have regarded the so-called "Elf-locks," plica or trica Polonica, as a part-phenomenon of weeping and impetiginous chronic eczema, wherein a diseased and changed condition of the hair is produced by spread of the inflammation from the skin to the hair-papillæ. This is evidently only a casual result, so that one must wonder at the importance attached thereto in the older text-books. At first it was actually looked on as an enzootic malady, with which at the same time was combined the idea of a "critical" sign. Its appearance was said to bring relief to an internal complaint; its removal to act fatally! The opinions as to its cause and importance were strangely at variance. But, as a matter of fact, it is an ailment of little moment in horses, a fact long since recognised in man, in whom it is known to be merely a weeping, chronic eczema of the skin of the head, complicated with felting of the hair.

**4. ECZEMA OF THE FLEXOR-SURFACES OF THE LOWER JOINTS  
OF THE EXTREMITIES.**

(*Eczematous Greasy Heels and Mallenders.*)

**Occurrence.**—Special names have always been given to localised ecsemata on the flexor surfaces of the joints of the extremities. The most important of these is the weeping eczema or eczematous greasy heels occurring on the pastern, which, in distinction to the pock-exanthema of horses, or so-called "dirt-mange," which also attacks the pastern, and the other surgical forms of greasy heels, is more exactly described as "gaping" or "dirt" greasy heels. Eczema on the flexor surfaces of the carpal and tarsal joints, or so-called mallenders, is much less common than greasy heels, because these joints are higher up, and therefore better protected against injurious material from the ground. Its course usually takes rather the form of chronic squamous eczema. For the other forms of greasy heels—the erysipelatous, gangrenous and verrucous—consult the text-books on surgery.

**Etiology.**—The flexor surfaces of the joints just named possess a predisposition to the development of eczema, owing to the displacement and formation of folds in the skin at every movement. To these is added the irritant action of dirt and dust and also of cold. On this latter ground eczematous greasy heels are observed more frequently in winter than in summer, and the hind-feet suffer oftener than the fore-feet, being more exposed to the action of dung when in the stable. Prietsch noticed the occurrence of greasy heels after sprinkling salt on the tram-lines in winter; but Siedamgrotzky, Born and others, maintain that such sprinkling, according to their observations, did no harm in this way. Clipping the fetlock and thus removing a natural protection for the pastern and its hollow, appears also to be an indirect cause of eczema by depriving the joint of that which should shield it against wet, cold and dirt.

The traumatic greasy heels noticed by Straub after exercising military horses in stubble fields has more resemblance to an infectious skin inflammation (erysipelatous greasy heels) than to a simple eczema. Its phenomena, which certainly much resemble those of greasy heels, were caused by a great number of small, superficial skin-wounds, with secondary erysipelatous and phlegmonous inflammation of the skin. A similar infective dermatitis develops also and not unfrequently in the further

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course of greasy-heels, when septic matter penetrates to the burst vesicles or into the cracks and fissures of the skin. Maisew has demonstrated that the streptococcus erysipelatis produces the erysipelatous, infectious form of greasy heels. But his idea that every case of greasy heels is erysipelatous in its character cannot be upheld.

**Symptoms.**—The lesions of mallenders and eczematous heels (considered by some authorities to be a form of grease) develop in a very typical manner. First the skin is red, swollen, painful and warmer to the touch (erythematous stage). But small blisters soon make their appearance (vesicular stage). These burst and discharge a watery, yellowish fluid which is at first odourless (moist stage). Owing to constant movements, the swollen skin forms thick folds, upon which fissures and cracks (so-called rhagades) readily form; these become inflamed and quickly covered with dry scabs. The hairs become stuck together, or they are bristly and readily fall out. The movement of the affected limb is stiff and awkward.

Although the lesions of mallenders and sallenders at this stage either heal or change to a squamous chronic form, with marked thickening of the skin and tendency to acute relapse, yet eczematous greasy heels, if long continued, may produce very serious changes in the fetlock. The secreted fluid decomposes upon the outer skin and macerates the epidermis. We then find a discoloured, greasy or pulpy secretion with a putrid smell, which, owing to its irritant properties, causes ulcerous loss of substance upon the skin, together with chronic thickening. Thus there arise upon the fetlock extensive sores covered with granulations which tend to become nodular or grapy (so-called "grapes"). The hair is at the same time erect and bristly. This thickening of the skin may, in the course of months and years, develop into true elephantiasis, in which case a chronic and permanent squamous eczema persists upon the outer skin (so-called squamous grease). Sometimes the leg is swollen as high as the elbow or thigh. "Thrush" is also a frequent accompaniment of chronic greasy heels. Finally, through the introduction of specific material, the eczematous greasy heels may develop into the erysipelatous and gangrenous forms.

**Therapeutics.**—The treatment of these conditions varies according to the stage of the disease. At first cleanliness and the application of a simple desiccant powder (such as oxide of zinc and starch) or powdered oak-bark are sufficient; some prefer

sinc or lead ointment. In later stages stronger measures must be adopted. First, one must endeavour to restrain secretion and decomposition of the discharge by desiccant, disinfectant and astringent remedies. For this purpose creolin and tar liniments are valuable, but desiccant powders (oak-bark, sulphate of iron, gypsum, charcoal) or astringent baths (solution of alum, sublimate baths, decoctions of oak-bark) are all very useful. Stages of medium severity may be healed by frequently applying a dry dressing or iodoform, sublimate, creolin or pyotannin. In cases of profuse granulation and induration, or elephantiasis, all remedies generally fail. A complete recovery to original soundness is here usually impossible, and our efforts must be confined to removal of the excessive granulations and indurations either by extirpation or by caustic remedies, that is to say, the treatment must be surgical, as in cases of new growths upon the skin.

#### C.—ECZEMATOUS SKIN DISEASES IN CATTLE.

**General Remarks.**—Generalised eczema is rarer in cattle than in horses, although nearly the same forms of eruption occur in both species. Both the papulo-vesicular and scaly forms of eczema have been observed in cattle; also an eruption attributed to hunger as well as an impetiginous eczema on the tip of the tail. In cattle, even more than in any other animals, eczema is usually traceable to errors in diet. The so-called "stalk disease," so much discussed in old text-books, is a proof of this. It presents at first a papular eruption, which later becomes pustular, and occurs generally on the extremities. It is caused by feeding on grape-stalks and vine-leaves. A similar eruption has also been noticed after feeding with maize-bran. But the influence of food seems to be much more important in that which, both practically and theoretically, is the most serious of all skin-diseases of cattle, viz., the so-called "malt-eczema," with which the grape-stalk sickness is obviously nearly akin. Compared with this disease, all other non-parasitic forms of eczema in cattle are so unimportant, that it suffices to refer our readers for details of their aspect and treatment to the conditions described in horses. Malt-eczema, on the other hand, demands detailed consideration.

#### MALT-ECZEMA (*Foot-rash*).

**Definition.**—Opinions still differ greatly on the nature

and definition of so-called malt-eczema of cattle. It is obvious that many diseases, which have etiologically nothing in common, and which as yet can only be clinically distinguished with difficulty, if indeed at all, have been grouped under this title. In veterinary literature, we find that the eruptions produced by feeding on brewers' grains and vintners' waste, on lucerne, white mustard or mouldy fodder, and even mange of the pastern and grease, have been confounded with malt-eczema of cattle.

The occurrence of grease in the ox has been denied by many observers; it is certainly very difficult to distinguish it clinically from malt-eczema, but the causes of the two conditions are quite distinct, for whereas the former is, as in the horse, usually associated with dirt and neglect, the condition which we are about to discuss is purely dietetic in origin.

The various hypotheses regarding the nature of malt-eczema will be discussed later. We shall consider the condition as a toxic exanthema analogous to mercurial eruptions as, for instance, produced by a hitherto unknown poisonous quality contained in potato waste and probably in the whole potato plant, tubers and stalks.

**Occurrence.**—True malt-eczema has only been known for about 80 years. Its first appearance in the twenties of last century is closely connected with the great extension of potato culture and the establishment of large distilleries for treatment of the products, with the consequent extended use of potato-mash for feeding purposes. Spinola first observed the disease in 1827, and more fully described it in 1836. Fattened oxen and male animals appear to suffer most, and after them, fattened cows. Milch-kine, on the contrary, often seem to escape. Certain animals have a pre-disposition to the complaint, while others, though fed on the same food, remain quite immune. The most serious outbreaks occur in spring and early summer, which is probably owing to the fact that germination in the potato reaches its climax about this time. Freshly stalled animals suffer most, so that the disease is most frequently found in byres where the cattle are often changed. Under such conditions cows only recently used for milking may, when newly stalled, also suffer. As a rule only the hind legs are affected from the foot to the hock. Sometimes the eruption is confined to the fore-feet, or again, all four extremities may suffer at once. Other parts of the body such as the trunk,

neck, shoulders, etc., may also be attacked, though this is rare. The condition of the byre has no influence upon the outbreak of the disease. It may occur in the cleanest and best regulated establishments, where straw is abundant and good, and the stall quite dry.

**Etiology and Pathogenesis.**—Malt-eczema is most frequently produced by feeding on potato-mash. Other mashes, such as rye and maize, are in this respect innocuous, as are also brewers' grains, although these have been blamed by some authors. The intensity of the disease is regulated by the quantity of mash consumed. Exclusive feeding on such mash, or the consumption of large quantities (16 to 18 gallons a day, per head) with but little dry food, is certain to produce it, whereas, if the quantity be more moderate (say 8 gallons) many animals will escape, whilst those which become affected will only suffer slightly. The gravest results arise from germinated potatoes, or from those in a state of advanced fermentation. But the singular fact must be mentioned that not every potato-mash is equally injurious. In different years the number of cases in the same byre varies considerably. This may be due to some difference in the fermenting process, i.e., in the products of fermentation, or to varied species or growths of potato, composition of the soil, kind of manuring, etc. It is possible that the effect of the potato may vary, like that of the lupin, according to the nature of the soil in which the plant has been grown. According to Baranski, the disease was first seen in Galicia after the introduction of the Gleason potato. The method of distillation seems to have some influence on the production of the disease, for malt-eczema principally appears after feeding with rapidly distilled mashes. Acidity of the mash does not appear to have any effect on the production of the disease.

The disease is not exclusively caused by feeding on malts, but may follow consumption of any part of the potato plant. Both raw and boiled potatoes (especially those which are sprouting or germinating), the water in which they have been boiled, and even the potato-tops, not unfrequently produce the condition. We are therefore forced to infer that the potato-plant itself contains some poisonous principle, which remains behind in the mash, and which, after extraction of the carbohydrates from the potato in the form of alcohol, is present in relatively larger proportion. Up to the present nothing

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certain is known of the nature of this poison. We may perhaps best form an idea of its action by assuming that on its excretion by means of the sebaceous and sweat glands of the skin, it exercises an irritant and inflammatory influence, and thus develops an eczema. This assumption is confirmed by the comparative immunity of milch-cows, in which the toxic agent is removed from the body by the very active secretion of the lacteal glands. The fact, moreover, that the milk of cows suffering from malt-eczema actually contains some poisonous property is proved by its injurious effect on calves and children, as frequently observed, the former being attacked by diarrhoea and the latter by eruptions. In the same way we explain the fact that animals suffer less if actively exercised, and the excretion of this matter be thereby stimulated and expedited. To this theory an objection has been raised in the question, why are, as a rule, only the hind feet affected? In reply we say that the eruption occurs at times in every part of the body. But we must bear in mind that the skin of the hind feet is especially predisposed to affection by the action of wet and dirt from the ground and of the dung, which, after feeding on mashes, is frequently very thin and even diarrhoeic. The smaller power of resistance to toxic exanthemata (medicinal exanthemata) possessed by the skin of the extremities we have already noted in a case of bromine eruption in a horse, which after strong doses of bromide of potassium had an eruption over the whole body, which was, however, only slight upon the trunk, but very intense towards the extremities.

## OTHER OPINIONS UPON THE NATURE OF MALT-ECZEMA.

1. The *solanin*, or *solanidin*, contained in potatoes has been blamed by some authors, and malt-eczema considered to be a form of solanin poisoning, an opinion confirmed by the fact that the amount of solanin in potatoes increases at the time of their germination, and that several cases of the disease also occur after feeding on germinating potatoes. But then the characteristic symptoms of solanin poisoning, which may, moreover, be experimentally produced, are absent in malt-eczema, viz., staggering, stupor, paralysis, etc. (see chapter on Solanin-poisoning, Vol. II., Chapter III., p. 210). Moreover, potatoes which are not germinating, and which therefore contain no solanin, may also produce the disease.

2. The *fusel oil* generated in alcoholic fermentation cannot, for many reasons, be accepted as a cause. It is present in every malt, for instance, in those of maize, barley, and rye, which produce no eruption. Besides, the potato mash is usually given entirely free from fusel oil. Further, there is no possibility of its presence in germinating potatoes or potato tops, and finally, the phenomena of fusel-oil poisoning are known to be quite different from those of malt disease.

3. The acids contained in the mash (acetic, lactic, butyric) are found in every other mash, whilst they are absent from potato-tops; moreover, their quantity is so variable that a harmless mash sometimes contains more than an injurious one.

4. John's hypothesis, which would bring the high proportion of potash salts in potatoes into etiological connection with malt-eczema, cannot explain the production of a vesicular eczema, because the potash-salts act as poison merely upon the muscles, and a large percentage of potash in food stuffs is not injurious.

5. According to Zürn the fermenting *barm-cells* contained in the yeast of alcohol (*saccharomyces cervisiae*) change in the bowels to rod-shaped organisms and, in combination with other bacteria and micrococci present in the intestines, produce a mycotic inflammation of the skin when brought into contact therewith through the dung. John has very properly raised the following objections to this theory. The same yeast fungi are present in all malts, and therefore every malt should produce this result, which is known not to be the case with malts of rye or maize. The incriminated barm-cells and fungi are found also in the dung of healthy animals fed on potato-mash, and indeed of such as have not been so fed, and must therefore be regarded as merely accidental ingredients of the dung. Then, too, malt-eczema may break out in the cleanest byre, and be absent in the foulest; and parts of the body are often affected which cannot come in contact with the dung. It has also never been shown that sound animals can be infected by the faeces of those suffering from the disease; attempts at inoculation have always miscarried, and indeed fomentations of warm mash have been employed with success against the disease. Baranski has used this very fact as an argument against Zürn's theory, as a result of his own experiments. Finally, there can be no question of the action of these *saccharomyces* in cases of malt-eczema following on consumption of raw potatoes, potato-tops, etc.

6. Rabe considers the disease to be symbiotic mange, and spread entirely by infection; that it is connected with the appearance and disappearance of the mite (*dermatophagus bovis*); and shows an intermittent character corresponding to the manner of life of this acarus. To this John rightly objects as follows: The mites are entirely accidental. He frequently found them on healthy animals, as well as after recovery from malt-eczema, whereas he only discovered them on one-third of those that were sick. Furthermore, tail-mange prevails in byres which have always been free from malt-eczema, without any affection of the fetlock or tibia, and without any other animals betraying signs of infection. Moreover, no attempt at inoculation with malt-eczema has ever yet succeeded. The disease can also at any time be cured by merely discontinuing the mash, and that without any further medicinal treatment. Further, tail mange—or rump mange—does not spread with such great rapidity over the whole body. Finally, the connection between malt-eczema and potato-mash is undeniable.

**Symptoms.**—The local phenomena usually commence about two or three weeks after first feeding on the mash, but earlier when such feeding is exclusive or abundant. There are at first redness and swelling of the skin of the pastern, in

consequence of which the animals begin to show stiffness and awkwardness in rising and moving their hind legs. Then vesicles appear upon the skin, which is very painful to the touch, and when these burst they become confluent and form large, moist sores, which dry up later and are often covered with very thick scabs and crusts. The hair is bristly, and the extremities of the affected limbs are more or less swollen. This eruption generally extends to the fetlock and may reach even as far as the hock or knee; occasionally in the male it extends along the inside of the thighs to the testicles, and in the female to the udder. In certain cases, not only the extremities, but the entire trunk, including the belly, breast, neck, back, etc., is covered with impetiginous scales. As a result the swollen skin becomes wrinkled, cracked and split, and discharges a purulent fluid which dries up into scabs. These local changes are accompanied by general disturbance. As a rule the sickness begins with a slight eruption-fever, lessened appetite, retarded evacuation, injection and even increased secretion of the conjunctiva, slavering, etc. Later, persistent diarrhoea often sets in, with rapid loss of strength and of flesh, which in extreme cases, and by no means rarely, leads to complete exhaustion and death. The cause of death then is either exhaustion, septicæmia or pyæmia. The percentage of fatalities is variable. Thus Baranski observed in Galicia a loss of 70 cattle out of 380, or nearly 20 per cent. In general the severest cases occur in dirty, ill-ventilated and badly kept byres, and in older animals, whose powers of resistance are small. Many animals are affected frequently, even from four to six times in a year. Recovery is therefore no guarantee of immunity. The course is, however, as a rule favourable, and recovery occurs in two or three weeks. The scabs gradually fall off, and the hair with them, leaving the skin bare. When neglected, however, the superficial vesicular and weeping eczema may be complicated by severe erysipelatous and phlegmonous, and even gangrenous, inflammation of the skin, which may in turn lead to septic and pyæmic infection (metastases in other organs). Sometimes a peculiar musty, mouldy smell is observed in byres where malt-eczema prevails.

**Differential Diagnosis.**—Malt-eczema may be confounded with various other diseases, the most important of which are:

1. *Grease.* This may produce an appearance clinically

identical with it, but is produced by an entirely different cause. It arises quite independently of feeding on mash, presumably from external causes. The changes produced by grease are also less severe, and we only perceive the erythematous and squamous stages. Yet vesicles also may undoubtedly be produced in grease. In practice one cannot go far wrong if one reckon every case of eczema following on a diet of potatoes or potato-mash as being malt-eczema, and all other non-parasitic eczemas of the feet as grease.

2. The *foot-scabies* caused by *dermatophagus mites*. Although we must admit that foot-scabies and malt eczema may be complicated one with the other, and that the presence of mange-mites on the hind-feet may cause a predisposition to the last-named disease, yet the clinical aspect and course of the two are different, apart from what is shown by the microscope. The eruption in symbiotic mange is decidedly slower and of milder course than that of the other condition, and only rarely attacks the lower extremities without spreading higher up also. Moreover, the character of the resultant eczema is mainly chronic and squamous, whereas malt-eczema presents chiefly an acute, vesicular or weeping condition.

3. *Foot-and-mouth disease* is also a vesicular exanthema, but it attacks the coronet and the interdigital space, is exceedingly contagious and arises independently of potato-food. Complications of the one with the other are naturally possible, but diagnosis should not be very difficult here.

4. *Panaritium*. An infectious inflammation of the terminal phalanges, not rare in cattle, and commencing at the coronet or interdigital cleft, with affection of the tendons, joints, periosteum and bones, can scarcely, as a purely surgical ailment of the hoofs, be confounded with a typical exanthema produced by malt.

5. Finally, the typical character of the eruption in eczema is not rarely so altered by manipulation on the part of the owner, his servants or of empirics (such as rubbing in petroleum or raw carbolic acid) that diagnosis may be somewhat difficult.

**Therapeutics.**—The simplest remedy consists in immediate discontinuance of potato-mash, or, if this be not possible, in reducing the daily quantity from 16 or 18 gallons to 4 or 8, and adding thereto a corresponding amount of dry or other food-stuff. Märker says that an addition of maize to the potato-mash will prevent malt-eczema, so long as not

more than 15 to 17 gallons of food be served daily, about one-third of which should be maize. Abundant exercise of the sick animal may also procure some relief. A great number of medicaments are in use. Rabe recommends a liniment of tar and soap (one part tar, two parts soap, 1 part flowers of sulphur, two parts alcohol), or 5 per cent. carbolic oil. Others use desiccant and astringent remedies in solution or in solid form, e.g., powdered oak-bark, decoctions of the same, lead-lotion, blue-stone, sugar of lead. We might also recommend the application of creolin or lysol in the form of a liniment (creolin and green soap of each 3 oz.; alcohol, 1½ oz. to 15 oz.); or of the ointment (5 to 10 per cent.) or of lysol or creolin, in a 2 per cent. watery solution. Of creolin spirits (4 to 10 per cent. solution). Finally, the greatest cleanliness in the byres is by no means unimportant. Although the origin of the disease has nothing to do with dirt, yet an excess of the latter may always produce a predisposition thereto, and aggravate otherwise slight cases.

#### D.—ECZEMATOUS SKIN DISEASES IN SHEEP.

**General Remarks.**—Non-parasitic skin affections in sheep are as yet little known and somewhat rare. According to Haubner-Siedamgrotzsky, a kind of rash, in reality a papular eczema, is occasionally met with which betrays its presence by tufting of the wool. A squamous eczema is also observed in the course of distomatosis and other cachectic affections, in which superficial layers of the epidermis scale off. In other instances this condition is traceable to neglect and bad attendance, and is then called "dirt-mange." May also speaks of a pruritic vesicular eruption as well as of impetiginous eczema under the names of "Prurigo" and "scabby-mange." Greasy legs likewise occurs among sheep, although the genuine malt-eczema has not yet been observed among them. Finally, pustular eruptions appear about the mouth after eating mouldy fodder.

The commonest and most important non-parasitic eczema among sheep is the so-called "rain-rot" (moist or "fatty" mange). In badly-nourished animals with open fleece the superficial layers of the epidermis become macerated during continuous rain or damp weather, and a weeping eczema develops under it, with swelling of the skin, formation of vesicles and scabs, loss of wool, itching, etc. It attacks especially

the back and loins, but may also spread forwards upon the shoulders, neck and head, and backwards to the root of the tail. Recovery usually follows spontaneously on cessation of the rainy weather, so that treatment is seldom necessary; but in severe cases the sheep must be brought under cover. Injury is generally restricted to falling-out of the wool.

#### E.—ECZEMATOUS SKIN DISEASES OF PIGS.

**Occurrence.**—Among non-parasitic eruptions in pigs the so-called "Rust" of young pigs ("pitchy-mange," "scale-eruption") is perhaps the most important. This presents at first a vesicular and later a pustular and scurfy eczema, which is most frequent in young and weakly or sickly animals, generally spreads over the whole body and may arise from a variety of causes (dirty stylos, accumulation and decomposition of the sebaceous matter of the skin, etc.). We usually first find vesicles, which quickly change into pustules and then dry up into thick, black, pitch-like scabs, beneath which a serous exudation takes place. In most cases this eruption is but a secondary symptom of some internal constitutional ailment, and may be compared to the scrofula of man. Treatment consists in cleanliness, frequent washings with soap, and good food. As the sarcoptic mange of pigs produces similar changes on the skin, it is also sometimes termed "rust." But it differs essentially in being contagious.

**Granular Eruption.**—Under this name Zschokke describes a peculiar chronic vesicular eruption in pigs, which occurs in patches the size of a hand on the ears, back, loins and root of the tail, and is marked by the greyish-violet colour of its blisters, which are very like the colour of small shot. The vesicles never burst and cause no itching (differing from rust). The process, says Zschokke, consists in a cone-shaped proliferation of the epidermis inwards, induced by specific micrococci, Ostertag and Johne regard this granular eruption as multiple dermoid cysts; while Lungershausen looks upon it as an arrested development (hypotrichosis). According to Olt, the ailment should be considered as a coccidiosis of the sweat glands, whereby the coccidia, after immigrating into the epithelium, provoke the formation of retention-cysts.

**Pityriasis Rosea.**—Schindelka thus names a skin-disease of pigs in which red and slightly raised spots about the size of a lentil appear on the breast and belly in somewhat circular form. These spots, at first round, have a diameter of  $\frac{1}{2}$  inch and seem to be made up entirely of small nodules. Later they grow flatter and paler in the middle, spread outwards, run one into the other and form sinuous lines. Duration, three weeks.

## URTICARIA, NETTLE-RASH, NETTLE ERUPTION, POMPHOUS ERUPTION.

**General Remarks on Definition and Causes.—**  
Urticaria is manifested by sharply defined, flat, raised swellings of the skin, the result of a quickly occurring serous transudation in the papillary bodies and the corium. It may therefore be regarded as a circumscribed oedema of the skin, or an acute localised serous dermatitis, which has, however, only reached the point of exudation of serum and not of infiltration with cells. The lesion of urticaria is clearly distinguished by this from a papule, and with this fact is connected also the possibility of the rapid disappearance of the rash by resorption of the serous fluid. The cause of this transudation of fluid is to be sought in a sudden dilatation of the capillaries in question, consequent on abnormal excitement of the peripheral vaso-motor nerves, or in an embolism. Urticaria may accordingly be looked upon as a vaso-motor neurosis or angio-neurosis, with abnormal permeability of the capillary walls. This affection of the vaso-motor nervous system may be due to skin irritation or may be of internal origin.

1. Among the *external causes* we may notice the action of irritant substances upon the skin, such as insect-bites, stinging nettles (*urtica urens* and *dioica*) and the hairs of caterpillars (*nethocampa processionea*). But other irritants may produce a nettle-rash on tender and predisposed skins, such as rubbing turpentine on a horse with colic.

2. *Internal causes* can only produce urticaria by disturbances in the innervation of the vaso-motor skin-nerves and only in animals which are predisposed. This predisposition is probably some disturbance of the vaso-motor nerve-system. Young, full-blooded and well-fed animals are especially subject to nettle-rash. The internal causes may also be presumed to be some irritant taken into the blood and which, granted the necessary predisposition, has a peculiar effect upon the skin. This seems to agree with the occurrence of urticaria during the course of several infectious diseases (petechial fever, strangles and the nettle-fever of swine), in which case the virus is responsible for the production of the inflammation. The exanthema of nettle-rash produced by certain disturbances in the intestinal canal (gastro and intestinal catarrh, icterus) must also be regarded in the same light, certain abnormal products of decomposition and digestion (toxines) are absorbed by the blood,

and excreted by the skin, producing inflammation. As in man, so too in animals, there is a certain idiosyncrasy or individual susceptibility to particular external influences, and especially to the action of certain kinds of food, which may be supposed to cause urticaria. Thus pod-fruits, buck-wheat, green potatoes, green oats or rapid changes in fodder are all blamed. In man, strawberries, raspberries, crabs, oysters and salt-water fish are supposed to produce the eruption on persons who are predisposed. Finally, nettle-rash may occur in the course of other complaints. Thus Schindelka observed it during the so-called Canadian or English horse-pox.

3. *Rapid cooling of the skin when the body has been over-heated* seems also to be not unfrequently an exciting cause of the eruption. It consequently occurs very often in spring and summer during great heat, in heavy torrential rain, after exhausting labour or rapid movement, as well as sometimes during rheumatic haemoglobinæmia. In all such cases it may be caused by retention or generation in the body of injurious matter as a result of the chill, rather than by any direct influence of the same upon the skin. And finally, in many cases of urticaria no assignable cause can be traced.

**Occurrence.**—From the foregoing it is evident that urticaria represents in by far the majority of cases a purely symptomatic exanthema. It ought, therefore, really to be only cited as an accompanying symptom of the particular disease with which it is associated. But the facts that nettle-rash is often the only visible phenomenon, and that the internal complaint cannot in all cases be surely demonstrated, decide us to discuss the eruption here, in doing which we shall only be following the usual practice.

The ailment is most common in horses, dogs and pigs, and next to them in cattle. Among 70,000 dogs taken to the Berlin dog-hospital during nine years, there were 108 cases, or 1.5 per thousand, of urticaria. On practical grounds it is advisable to treat the complaint in horses, dogs and cattle all together.

#### I.—URTICARIA IN HORSES, DOGS AND CATTLE.

**Symptoms.**—The urticaria of horses, dogs and cattle, formerly known as "heat-boils," or "boil-fever," is characterised by an extraordinarily rapid appearance of pomphi, which in the course of a few hours or overnight will spread over quite a

large area. At first there are isolated patches of soft swellings the size of a pea, above which the hair looks rough, and which appear mostly upon both sides of the neck, round the shoulders, along the back, sides and buttocks. But these multiply and grow quickly, very often coalesce and then form patches as large as the hand or a dinner-plate, when even the head, fore-legs, lower thighs and udder may be attacked, giving the poor animal a very unsightly appearance. Pruritis is only occasionally present. Pug-dogs seem to be most readily affected.

Sometimes the pomphi are also met with upon the visible mucous membranes, as of the nose, mouth and vagina. Their presence in the nose may lead to disturbance of breathing (Schleg). Zipperlen found the conjunctiva in a cow very much swollen, and the mucous membranes of the rectum and vagina pressed out in thick rolls. Moreover, if the transudation penetrate between the rete Malpighi and the epidermis, vesicles may develop upon the pomphi (so-called nettle-blisters, pomphosis); these then usually produce intense itching and loss of hair. In addition to these processes upon the skin and mucous membranes, various disturbances of general condition are perceived, according to the nature of the chief causal malady. Very often symptoms of slight fever—seldom of high fever—are an accompaniment. The differentiation between nettle-fever and simple feverless nettle-rash, formerly based on this fact, is consequently of no avail. Weariness, depression and disinclination for work are associated with the condition, or signs are seen of gastro-intestinal catarrh, loss of appetite, diarrhoea, constipation or slight jaundice. In one case of a horse, temporary outbreaks of sweating were noticed alongside the urticaria. Usually the eruption vanishes as quickly as it appears, say in one to two days. But fresh attacks often recur, which make the course longer and even chronic. Some animals indeed suffer several times in one year from urticaria.

**Therapeutics.**—Treatment is generally superfluous; in many cases fasting and warm covering suffice. If there is evidence of gastric disturbance, attention to the bowels may be advisable; give purgatives, such as tartar emetic, aloes, calomel, or, as a milder evacuant, Epsom or Glauber's salts.

## 2. URTICARIA IN PIGS (*Nettle-fever, Brick-rash*).

**Definition.**—The nettle-fever of swine, "Brick-rash" (Danish nodular erysipelas), was in earlier decades regarded

as a milder form of erysipelas. Following Haubner's example, it has, on practical grounds, been separated therefrom and treated as an independent disease. In later years nettle-fever has again been indicated as a complaint allied to the erysipelas of swine, and this as the result of bacteriological investigations (Jensen, Lorenz and others). According to these, the erysipelas of pigs runs sometimes benignly as nettle-fever, and sometimes malignantly as true swine erysipelas, again as a necrotic inflammation of the skin, or furthermore as a chronic endocarditis, etc.

If these statements are confirmed, then we must assume that, like other infective diseases, swine erysipelas may run a benign course as an exanthema possessing the aspect of urticaria; and that many cases of so-called nettle-fever must be reckoned as erysipelas. On the other hand, it is quite certain that, as in other domestic animals, so also in the pig cases of urticaria occur which must be due to unknown causes, such as gastric disturbance, and quite independent of the disease caused by bacilli. There is, therefore, no conclusive reason why we should omit the consideration of nettle-fever from this chapter. Rather would we differentiate between :

1. An *independent, non-infectious* urticaria ; and
2. A *symptomatic* urticaria, which is presumably produced by *bacillus-erysipelas* (comp. chapter on Swine Erysipelas, Vol. I., p. 70).

**Symptoms.**—The phenomena of non-infective urticaria (which usually appears sporadically) consist in a rapidly appearing eruption of pomphi on the skin, which vanishes just as quickly and is accompanied by gastric and feverish disturbances. On animals previously quite sound there appear, and usually overnight, a number of pomphi, about the size of a hazel nut, which are painful when touched, rise  $\frac{1}{2}$  to  $\frac{1}{4}$  inch above the surface, and are about the area of a crown-piece, but by the confluence of several may become as large as the palm of the hand. They appear mostly on the upper trunk, breast, loins, upper thighs and belly. These pomphi vary in colour according to the stage and intensity of the condition. At first they are merely red spots, rising a little above the surface, whereas in the advanced stage they are white elevations with a red margin (so-called white nettle). Sometimes a serous exudation is also to be seen on the top of the pomphi, which dries up to a scab and is cast off. The general symptoms are lessened

appetite, as well as an invariable constipation and at times also vomiting. The animals seem melancholy and dull, bury themselves in their straw, are difficult to bring to their feet, show strongly injected mucous membranes, accelerated breathing and medium fever (about 104° Fahr.). In a few cases the gait is stiff and awkward, which points to some rheumatic affection of the muscles.

**Prognosis.**—In contrast to erysipelatous urticaria, nettle-rash (which is usually not infective) follows an exceedingly benign course. In slighter cases recovery follows in one or two days, in severer ones, after four to six at most. No fatal case has yet been known. A turn for the better is indicated by abatement of the constipation; at the same time the exanthema vanishes, the pomphii grow paler, become soft and withered and then quickly disappear. And so the attack comes to an end.

**Treatment.**—Owing to their benign aspect and typical course, ordinary cases of nettle-fever require no treatment, or merely dietetic. To relieve the constipation one should administer clysters, soap-enemas, etc., according to symptoms, and also give doses—not too small—of calomel (15–60 grains). Haubner recommends saltpetre ( $\frac{1}{2}$  dram) with glauber salts ( $1\frac{1}{2}$  oz.); with honey for licking or as electuary. One may also reduce the inflammation of the skin by frequent douches of cold water.

#### GANGRENOUS DERMATITIS.

(*Gangrene of the white marks.*)

**Etiology.**—The causes of gangrenous dermatitis in horses and cattle, which specially attacks piebalds and animals with white markings, and is occasionally enzootic in its spread, must be sought exclusively in the action of external irritants. As a result of intensified erythematous and eczematous inflammation, the affected portions of skin ultimately become gangrenous. These irritants are of different kinds. I. The intense power of the sun, or of a hot, dry wind, may in many cases be regarded as the direct cause of this condition (so-called sun-gangrene, *gangræna solare*). This represents a higher degree of erythema solare. According to Plassio it is noticed in Africa in horses with unpigmented skin upon the parts unprotected

by saddle or harness. 2. The fact that the skin is *unpigmented* causes a *pre-disposition* not only to the action of the sun, but also to that of other irritants, as lacking the protection of the pigment. Consequently the white marks on the head and extremities and the light spots on piebalds are most readily affected. 3. Certain *fungi*, it is evident, are also able to produce a gangrenous dermatitis in many cases, viz., in the so-called clover-sickness, and perhaps also in lupinosis, and this occurs especially on the white parts of the skin. Spoilt vetch and green food covered with mildew (*erysibe fungi*), as well as plant-lice, have been charged with causing this inflammation. 4. The so-called gangrenous or discharging greasy heels are due either to an increase of the irritation usually present in greasy heels (severe frost, etc.), or to some infective process.

With regard to the dry, diffuse gangrene of the skin in pigs, see the chapter on Swine Erysipelas (Vol. I., p. 70, etc.); and for mummification of the extremities see that on Poisoning by Ergot of Rye (Vol. II., Chap. III., p. 202).

**Symptoms.**—Gangrenous inflammation of the skin begins with the appearance of erythema or eczema. The swelling of the skin rapidly increases, cracks and fissures are formed and necrosis of certain patches quickly sets in, which is sometimes only superficial and at others very deep. In the former case leathery scabs are formed upon the skin; and in the latter the whole skin shrivels up like parchment right down to the subcutis, and is all thrown off by suppuration. This casting-off of the necrotic parts takes place with formation of a line of demarcation. Abscesses and fistulae may also be developed. Healing is produced by granulation and formation of scabs.

**Therapeutics.**—Treatment consists at the outset in rubbing in lead or lead and tannin ointment, carbolic oil, carbolic ointment, iodoform salve or creolin ointment, creolin-liniment, creolin-water, and in the application of pyoktanin, etc. Later, i.e., after necrosis of the skin, the necrotic portions should be removed by knife or scissors and an antiseptic dressing applied.

#### BULLOUS DERMATITIS, VESICULAR ERUPTION, PEMPHIGUS.

**Definition.**—In bullous dermatitis we have to do with a formation of large blisters, as large sometimes as a hen's

egg or the palm of the hand, rising upon an intact or reddened skin, whereas in eczema only small blisters occur. Nothing positive is known as to the cause of this singular exanthema in our domestic animals.

**Symptoms.**—Various reports on the phenon have been published. As a rule the course of the disease is acute (*pemphigus acutus*). Loiset saw the eruption appear ensoötically in cattle. Large, round or oval blisters, as much as four inches in circumference, showed upon the loins, croup and thighs, they were charged with a clear fluid and, soon bursting, left behind them wet places, which scabbed over and then healed with desquamation of the skin, leaving shiny patches. Seaman noticed a very similar eruption in an ox, which was accompanied by feverish shiverings, and in which the blisters were the size of a hen's egg. Lucet discovered the streptococcus pyogenes in the blisters on a cow. Dieckerhoff observed the eruption on five horses. On the belly, head, neck, thorax and between the thighs, blisters, from a walnut to the palm of the hand in size, arose upon the skin, full of a fluid, either clear or of a yellowish tinge. Usually the uplifted epidermis broke, leaving large sores, from which oozed a fluid secretion. In addition to sharp itching, there was general disturbance of condition. Similar cases have been observed in horses, mules, pigs and dogs. One instance of chronic pemphigus watched by Fröhner proved fatal after lasting some weeks. There were numerous watery blisters and ulcers all over the body accompanied by great emaciation.

This bullous dermatitis has a great resemblance to the vesicular eruption in man known as pemphigus, the etiology of which is also still very obscure. The latter, as seen in man, occurs sometimes as an acute infective disease, and then a vaso-motor neurosis, and again a vegetable parasitic disease of the skin. The above-mentioned cases in cattle seem to be especially like the *pemphigus vulgaris* of man, which also runs a benign course; whereas *pemphigus foliaceus* finally leads to death (compare the above case in a dog).

**Herpes labialis.**—In addition to this bullous dermatitis characterized by large blisters, some authors have described a specific vesicular eruption (especially in horses) upon the lips and nose, which they call "heat-eruption," or "herpes labialis." The exanthema is characterized by small, quickly-developed blisters, grouped together in clusters, which attain the size of a lentil, contain a clear fluid, and soon heal with forma-

tion of scab. It must be admitted that this specific eruption much resembles the herpes facialis and genitalis of man, and so far agrees with it that it occurs in the neighbourhood of the natural orifices of the body. But so much misuse has been made of the term "herpes" in animal pathology that it would certainly be more appropriate, following Garisch's suggestion, to restrict its use to a specific parastic skin disease, viz., herpes tonsurans. With the herpes zoster (girdle-erysipela) of man which appears in the course of a nervous complaint, the young example described above as a specific vesicular eruption has certainly nothing in common.

#### LOSS OF HAIR AND WOOL (*Alopecia*).

**Occurrence.**—The falling out of hair and wool occurs as an independent skin affection, apart from other skin diseases, sometimes locally and sometimes over the whole body, being known in the former case as area celsi. The nature of alopecia must be sought in certain disturbances in the nutrition of the skin (tropho-neurosis) which cause atrophy of the hair-roots. The co-operation of parasites cannot be absolutely disproved, though up to the present not clearly established.

**Etiology.**—The causes of alopecia are partly known. A general falling out of the hair presents obviously a symptom of some general disturbance of nutrition and is therefore termed *alopecia symptomatica*. It is noted where the food conditions are bad; as a result of scanty feeding, and on the contrary in great obesity; during pregnancy; during suckling; in the course of prolonged consumptive disease; during certain blood disorders, such as petechial fever; after protracted sweating; after certain interruptions in the circulation of the skin; as an inherited and congenital ailment (*alopecia congenita*); after eating certain kinds of food, such as sour hay from swampy soil; or after psychical excitement. The *area celsi* (*alopecia areata*) is regarded by some as a tropho-neurosis, that is, a disturbance of nutrition in the region of the skin-nerves, and by others as a parasitic affection.

**Symptoms.**—In *alopecia symptomatica* a general dropping out of hair occurs all over the body, after which the animals appear perfectly naked. In horses the alopecia is sometimes preceded by gastric disturbance, and also by oedematous swellings on the limbs, lower belly and lower breast. In other cases no accompanying signs can be perceived. As a rule, the hair grows again in a few weeks. From our observations

alopecia in dogs is very common, especially upon the back. In Berlin 224 cases (3 per 1,000) were observed among 70,000 cases treated in the dog-hospital in nine years. A darker pigmentation of the bare surfaces is usually noticed as an accompaniment, which probably acts as a protective substitute for the hair. Siedamgrotzky found on examining such pigmented spaces under the microscope that, in addition to atrophy of the hair and hair-bulb, the cells of the rete Malpighi were quite infiltrated with black and brown granules; the same also in the root-sheaths of the hair-follicles and the gland-cells of the sebaceous glands. From sheep the wool usually falls out in coherent masses, leaving large naked patches.

2. *Alopecia areata*.—This shows itself at first in small, round, bald spots, which gradually grow larger. It is commonest in dogs and resembles very much the appearance of herpes tonsurans, only that the fungus characteristic of the latter cannot be found. Röll noted a case of a horse in which the hair came off at first only on very small spots, but wherein the alopecia spread in the course of a year over almost the entire skin. Here also abundant black pigmentation was present along with atrophy of the hair-bulbs. No micro-organisms could be found, so that some atrophic process in the skin must be assumed as the cause. The complaint is very obstinate and defies all treatment.

**Therapeutics.**—There are several stimulating remedies which may be tried to promote regrowth of the hair in alopecia. Spirit is used most frequently, or spirit of soap and camphor. Also tincture of cantharides, if sufficiently diluted, may be rubbed on (1 : 5 spirit, or with fat as a pomade). In alopecia areata we recommend, in addition to the anti-parasitic Peruvian balsam (1 : 10 spirit), creolin (1 : 10-20 spirit) and tincture of iodine mixed with equal quantity of spirits of wine and laid on with a brush for several days successively.

**Trichorrhexis nodosa**.—A peculiar affection of the hair, evidently identical with that described by Kaposi under this name in man, has been observed by Trofimo. Among nearly two-thirds of the horses in an artillery brigade symmetrical spots appeared on the back, loins and croup, on which the hairs were all broken off about a third of an inch from their root, or swollen out into a nodule. The place of fracture was found under the microscope to be considerably fibrillated. A similar interesting case is described by Mégnin. This hair-affection seems to be common in the Prussian army, and in one year was found in eight regiments. According to Steinhardt's description, small, greyish-white nodules appear upon

the diseased hair at distances of from  $\frac{1}{2}$  to  $\frac{1}{4}$  inch and progressing from the tip of the hair to the root. At these nodules the hair bends or breaks off. Both the protective hair (mane and tail) and the covering hair are affected. Upon the latter irregular patches of such broken hair are found about the size of a crown piece. Treatment with sublimate, creolin, carbolic acid, tar, ichthyol, etc., is fruitless, and the hair is not renewed until the change of coat. According to Kalkoff and Seegert, a daily washing of the affected places with a five per cent. watery solution of pyrogallol was found effective. It is thought that the complaint is transferred to other horses by the grooming utensils (also in the beards of the attendants).

**HÆMATIDROSIS. BLEEDING OF THE SKIN (so-called Bloody Sweat).**

**Etiology.**—The numerous cases of bleeding of the skin described in veterinary literature under the name of "bloody sweat" have a very obscure etiology. Many of them are probably purely symptomatic haemorrhages occurring in the course of severe general disease, such as petechial fever, anthrax, septicæmia or scurvy. Others belong to the realm of acute exanthemata, which may sometimes be complicated with haemorrhage. But if we omit these, there still remain a number of quite peculiar cases of bleeding of the skin, which have not arisen through secretion by the sweat-glands, as their false designation, "bloody sweat," might lead one to suppose, but represent haemorrhages either by rheins or by diapedesis. A definite cause for this cannot be discovered, but probably a general disturbance of nutrition of the vessels lies at the base of it. To this class belong especially the skin-haemorrhages of cattle. Besides these haemorrhages there are certain bleedings of the skin which occur particularly among thoroughbreds and horses of oriental, Hungarian, Russian and Tartar breed, which are harmless in their nature and were aforetime considered to be nature's self-aids. Probably these latter arise from the settlement of nematodes in the skin (*filaria haemorrhagica*) and from the vascular injuries thereby effected (*haematidrosis parasitaria*). In fact, says Dronilly, such skin-bleeding does actually occur in Hungarian horses, being of parasitic origin and caused by *filaria haemorrhagica*.

**Symptoms.**—The extravasation of blood occurs in drops and in various parts of the body. In slighter and benign cases it only affects the skin, and that most commonly on the shoulders, neck, breast and extremities. In those cases the bleeding is

slight and soon abates. If, however, a general haemorrhagic diathesis be present, then we have haemorrhage into the mucous membrane, and bleeding occurs from the nose and intestines, we see bloody milk and bloody urine. From this severe anaemia often develops, of which the animal not rarely dies. General disturbances such as slight fever, gastric disorder, etc. also usually accompany this last kind of haemorrhage. In horses of pure blood the bleeding occurs sometimes in a stream for a brief period.

**Therapeutics.**—A treatment of partial skin-haemorrhage in thoroughbreds is seldom necessary; at most one may wash the bleeding places with cold water, or try anti-parasitic remedies. But for general haemorrhagic diathesis an internal styptic treatment must be adopted, viz., administration of ergot, tannin, sugar of lead, etc.

#### ACNE AND FURUNCULOSIS.

**Definition and Causes.**—Under the name of acne we understand a suppurative inflammation of the follicles of the skin (suppurative folliculitis), which has arisen in consequence of accumulation and decomposition of the matter secreted by the sebaceous glands. The provocation to such inflammation may come either from without, or from the blood and penetrate to the sebaceous glands and follicles. The skin-affection is local but deep, and reveals itself either in papules or pustules. A typical form of acne arises from incursions of the acarus folliculorum into the follicles (a pustular form of acarus eruption). In contrast to this parasitic acne, which will be discussed elsewhere, the common acne is a non-parasitic or non-infective skin disease, which is oftenest seen in dogs on the head where the muzzle fits, as a consequence of persistent irritation. Acne also occurs in horses and usually under the harness (neck, shoulders, back), and also in Negretti sheep on the inner surfaces of thighs and abdomen.

By the name of furuncle we describe large acne pustules which cause necrosis of the follicles (gangrenous, suppurative folliculitis). It is therefore a development of acne and represents only a higher degree of follicular inflammation.

**Symptoms.**—The acne of horses has been described under many names, such as nodular, tubercular, pimply rash,

knotty scabies, heat-nodules, tallow-nodules, pustular measles, callous tubercles. But many of these skin diseases do not belong to true acne, but to papulo-vesicular eczema (comp. p. 419), from which it is not always easy clinically to distinguish. It is most frequently found under the seat of the saddle as small, firm and painful nodules, from which little tallowy or pus-like plugs may be squeezed, and on whose summit a yellow-brown exudation or dry scab may often be found. It arises after severe exertion from the prolonged irritation of the saddle and consequent decomposition of the skin-secretions, with subsequent infection of the sebaceous glands. Similar nodules, though generally somewhat harder and larger, up to the size of a pea, are often found beneath the harness. After the tallow or pus-plug is pressed out, cup-shaped ulcers remain with very narrow entrances with jagged edges. In general the acne-nodules on horses are distinguished by the fact that their contents consist less of pus than of a tallowy, greasy matter like groats. The surrounding skin sometimes shows signs of chronic indurating dermatitis (sclerosis), as well as of eczema squamosum. Contrary to papulo-vesicular eczema, we seldom find acne spread over the whole body, but rather confined to the above-named spots.

2. In dogs the disease is generally localised upon the skin at the back of the nose. It usually occurs in bull-dogs, mastiffs, pointers and Leonberg dogs, that is, in those with a long fore-head. The pressure of the muzzle causes at first a superficial inflammation, which spreads to the more deeply seated follicles, causing suppuration with a swelling-up of their orifices. The skin feels thick and hard and, on pressure, discharges at various points a bloody or purulent matter. Nodules the size of a lentil, pea or even a hazel-nut are also present, with a sunken purulent centre. In addition to the back of the nose, we find acne-nodules and pustules, or the furuncles produced thereby, upon the lips, forehead and temples. In rarer cases furunculosis occurs upon the skin of the trunk and extremities, in patches as large as a five-shilling piece or the palm of the hand, which are interspersed with nodules and pustules from which a bloody-purulent secretion can be squeezed. Sometimes the pustules run together into large abscesses, above which the skin has a bluish red or violet colour. This form of furunculosis we find mostly in griffons and long-haired pointers. Its causes are unknown, and are perhaps to be sought in a congenital difference of structure in the sebaceous glands. When found on the trunk

or extremities the prognosis is very unfavourable, as it always spreads and may take months to heal. The complaint is much more frequent among the dogs of large towns, owing to the muzzling orders often in force there. Of 70,000 treated in the Berlin dog-hospital in nine years, 452 (or 0.7 per cent.) suffered from a high degree of acne and furunculosis.

**Therapeutics.**—The treatment of acne and furunculosis is mainly surgical, consisting in pressing out, opening and cleaning the abscess-foci. Then disinfectants must be introduced into the open follicles, for which we have found an ointment made of Peruvian balsam and creolin (1 : 10) especially useful on horses and dogs. Cauterization of the hollows with a nitrate-of-silver pencil is also very advisable. In very obstinate cases the only remedy is total extirpation of the affected portion of skin. As a prophylactic we recommend avoidance of all external irritants. In furunculosis of the ridge of the nose the muzzle must be kept off for a time, or at least lined with soft felt, to prevent relapse. Slighter attacks of acne in horses heal of themselves on removal of their exciting cause.

The remaining anomalies of the skin belong partly to the department of general pathology, such, e.g., as excessive sweating (hyperidrosis), extreme secretion by the sebaceous glands (seborrhœa), the so-called hardness of skin, sclerodermia of swine, anomalies in the change of coat and formation of pigment; or else they are purely of surgical character, such as warts, horny skin, elephantiasis, ichthyosis, phlegmona, erysipelas, etc.

## II.—VEGETABLE-PARASITIC SKIN DISEASES (*Dermato-mycoses*).

**General Remarks.**—Of skin diseases caused by vegetable parasites on our domestic animals only three have so far been positively demonstrated: 1. Ringworm (*tinea tonsurans*); 2. Honey-comb scab (*favus*); and 3. Dermatitis pustulosa contagiosa canadensis (the so-called English horse-pox). Of these the two first are by far the most important. The question has often been raised whether they are caused by two distinct fungi or by two variations of one and the same fungus. The results of microscopical investigation indicate a unity of fungus species, as no essential difference can be found between the hyphae and conidia in *tinea tonsurans* and *favus*. But the later studies of Grawitz, who at first accepted the identity of the two fungi with each other, and also with the *oidium albicans*, have now proved that *tinea tonsurans* and *favus* are produced by two separate species of

fungus. It is true the spores of both are very similar, but they show many differences in growth, liquefaction of gelatine, form of cultivation and conditions of fructification. Thus in the microscopic aspect of favus the mycelium predominates, while in tonsurans the spores are most numerous. Oidium lactis has nothing in common with them. According to Quincke, favus alone may be produced by three distinct fungi. A further argument in favour of their being two different dermatomycoses lies in the fact that the clinical aspect of both is quite different, and that intermediate forms are usually absent. On this latter ground alone we must rank tinea tonsurans and favus as two essentially distinct skin affections.

Besides the three dermatomycoses just named, other skin diseases have been described in which fungi have been found which could not be more nearly defined. Thus Siedamgrotzky reported one clinically much like herpes circinnatus, in which, however, the presence of trichophyton could not be proved. Similar observations we have ourselves often made with dogs. Leisering describes an eruption producing baldness round about the orifice of the cloaca in a cock, which was transmitted by copulation to the hens, upon whom greenish-yellow conidia were found. He and Engel have also reported on several vegetable-parasitic affections of the root of the tail and its hair in horses.

#### TINEA TONSURANS (*Bald Scab. Ringworm*).

**Etiology.**—The various diseases known as ring-worm, depillary dry scab, scab-mange (dermatomycosis tonsurans, tinea or porrigo decalvans, tinea scutellata), and in calves as dough-mouth or doughy evil, doughy mange and mouth-mange, in other animals as lamb-mange and goat-mange, only merit their title of "mange" in so far that they are all caused by a vegetable parasite. This fungus, which is called trichophyton tonsurans, belongs to the hyphomycetes and was discovered by Malmsten in 1845. As the cause of ringworm in our domestic animals it was first identified by Gerlach, who in 1857 published a classical work on the mange of cattle. The transfer of the fungus to other animals occurs through the combing and brushing apparatus, harness, covers, or by contact with neighbours or, in sucklings, during sucking. Bulls in particular may transmit the affection to a whole herd. During pastureage it may also be easily spread, as here the animals come much in contact.

Cattle are most readily affected, both older animals and especially young ones and sucking calves; after them come

dogs. Horses, goats and cats suffer more rarely; pigs and sheep more rarely than any. The complaint can, moreover, be transmitted to rabbits by inoculation. Ringworm is in general not a common or wide-spread skin affection, but seems rather to be located in certain byres and districts; and also in certain countries (England, Holland, France, Switzerland) is commoner than in others.

Many cases have been described in veterinary literature in which the tinea tonsurans of animals (cattle, dogs, horses) has been transmitted to man. Those affected have usually been the stable attendants who have been infected when cleaning or milking the animals, or rubbing them with ointment; or this has occurred in skinning them after slaughtering. Dogs seem to transmit the disease especially when being fondled. This infection in man may assume epidemic proportions; thus in 1840 most of the inhabitants of the Swiss village, Andelfingen, were infected by sick cattle. Köhler reports a case in which a newly stalled calf infected 21 cows, 5 calves, 9 horses, the herdsman and several other persons.

**Morphology and Physiology of the Trichophyton Tonsurans.**—Microscopical investigation of trichophyton tonsurans enables us to recognize two elementary forms, viz., threads (*hyphæ*) and spores.

1. The *threads* or *hyphæ* are tender, elongated tubes, about four micro-millimetres thick, straight or undulated, dichotomous and possessing simple contours, sometimes articulated and sometimes not. They twist round the hair like nets, penetrate it and, when present in large numbers, form a perfect layer or bed of fungi (so-called mycelium).

2. The *spores* or *contidæ* are small, spherical or elliptical cells, very refractile, and sharply defined, of about the same diameter as the hyphæ, from which after segmentation they develop. They are sometimes ranged side by side like a string of beads, and are usually much more numerous than the hyphæ, so that the latter are scarcely visible. These spores are very resistant and retain their power of germinating for more than a year.

The fungus vegetates especially in the hair-follicles and in the hair itself. The lower part of the hair-stalk is first enveloped in the fungoid mass, when a white, cylindrical mantle may be perceived by the naked eye at the base of the hair as a wing-shaped accretion. Later the fungus penetrates to the hair

sheaths and roots, producing inflammation in the follicles which finally results in loosening and loss of the hair; Lastly it lodges in the interior of the hair itself, splitting it into fibres and rendering it brittle. The interior of the hair is often found so interpenetrated with conidia that nothing more of it is to be seen. The fungus seems to flourish best on dark skins and dark hair. The respective diameters of the spores and hyphae are not the same in all animals, indeed on the same kind of animal they may vary considerably. Mégnin believed, therefore, that there must be two species, viz., a *trichophyton tonsurans* of horses and a *trichophyton epilans* of cattle; but differences in size are obviously of secondary importance and probably depend on variations in the source of nourishment. According to Grawitz, the tinea fungus liquefies gelatine after dissemination of seed more quickly than the favus fungus. When placed upon agar-agar, *trichophyton* produces a green turf-like covering, while the favus culture shows a stellate vegetation.

**Symptoms.**—In describing the morbid aspects of ring-worm we must first remark that a uniform description, fitting all cases, cannot be given, because the symptoms vary most remarkably according to the kind of animal and its breed, nay, even in the same animal, with the position and age of the eruption, and also according to its having been further affected by scratching, gnawing, rubbing, etc. In general we may cite as characteristic signs the following:—In the beginning clearly defined, round places, either hairless or studded with broken hairs, and mostly situated on the head, neck and extremities. These, which are at first the size of a lentil, extend outwardly and may attain the size of a crown-piece or more. The patches are mostly scattered, or they run together and form larger ones. Sometimes the process ceases in the middle while spreading from the outer edge (so-called ring-worm, *herpes circinnatus*). Not unfrequently the eruption spreads over much of the body or over its entire surface, producing complete nakedness. On examining under the microscope hairs which have fallen or been pulled out, we find the hyphae and conidia described above. The upper surface of the hairless patches shows either no inflammation at all, or else very varying degrees of it. With thick and more resistant hair, such as that of the horse, we find increased desquamation of the epidermis in the form of ash-grey, slaty or asbestos-like scales (*tinea tonsurans*

maculosus). On finer skins we observe at first hyperæmia and swelling, in consequence of which the affected skin projects above the surface. Later on, vesicles are formed (*tinea tonsurans vesiculosus*), which quickly burst and are dried up to thick crusts and scabs, which in full-grown cattle are of different colours and leathery consistence, but in calves are a whitish grey colour like that of bread-dough, and also much fissured. The nature of these scabs is regulated by that of the hairy coat; being very thick where the coat also is thick, so that, as in cattle, they may be  $\frac{1}{2}$  inch thick, while on parts less densely covered they are very thin. Beneath the scab is a suppurating surface, and after the crust has fallen off the places may heal of themselves.

The aspect of the disease in various kinds of animals is briefly as follows:—

**I. *Tinea Tonsurans* in Cattle.**—Small, round, sharply defined spots are found, covered with scabs or scales, standing either singly or in groups and usually upon the head and neck, more rarely scattered over the whole body. These project more or less above the skin and are either bald or covered with greyish white scales, or with bristly hair. They may attain the size of the palm of the hand, and sometimes run into each other. On its outbreak, and also during the healing process, the eruption produces itching. The grey-white, asbestos-like scabs may become  $\frac{1}{2}$  in. thick upon black skins; but on white ones they are thinner and more yellowish. Underneath we find a sticky, purulent fluid, as well as small and ulcerous hollows, which represent the distended, emptied and hairless follicles. Healing usually proceeds under the scabs, which gradually fall off, leaving small, slightly scurfy spots, upon which the hair slowly grows again. In other cases the skin remains very much thickened and arranged in folds (*elephantiasis*). The eruption lasts from six to twelve weeks. If, however, the normal course be disturbed by external influences, rubbing or scratching, fresh outbreaks occur within the healed patches, whereby, to some extent, a new inoculation takes place, and the ailment may be prolonged for six or twelve months.

In sucking calves we find the eruption chiefly about the mouth—so-called dough-mouth or mouth-mange. This affection, which is seen also in other sucking animals (lambs, kids, pigs) belongs probably in its essentials to *tinea tonsurans*, though it must be granted that a non-parasitic, impetiginous *eruption* may also occur which is confined to the region of the

mouth. But in most cases the evident contagiousness of the disease, as well as its clinical aspect, show it to be tinea tonsurans. Hahn has shown by his microscopical investigations into the nature of "doughy mange," that it is caused by tinea tonsurans, and published a description and drawing of the fungus. The phenomena of the eruption he describes as consisting in impetiginous round deposits upon the edges of the lips,  $\frac{1}{2}$  to 1 inch in size, lying on the visible surface and occasionally spread over the whole body, chiefly consisting of dry, bran-like, scaly matter, through which the hair projects, the latter being split up into fibres and destroyed by the fungus. In young animals the complaint is very obstinate.

2. **Tinea Tonsurans in Dogs.**—This disease chiefly affects the head and extremities of dogs, but not rarely spreads also over the whole body. It begins with round, sharply defined and solitary bald spots. Later we find isolated, clearly defined, hairless patches of round or oval shape and from  $\frac{1}{2}$  to 1 inch across. These are most numerous on the head around the lips and eyes and upon the extremities; not unfrequently they combine to form larger lobulated areas. According to the locality and age of the process we perceive quite varied morbid signs upon these patches, which are either quite bare or covered with solitary hair-stumps. Very often the skin shows no symptoms of inflammation; and the tinea tonsurans occurs then with the aspect of alopecia or area celsi. But the spots are just as often incrusted with asbestos-like and dirty grey scales; in other cases with thicker scabs, by which the lower parts of the hair are stuck together. Beneath these scales the skin is from copper-red to brownish-red in colour and covered with numerous granules the size of a millet-grain (swollen hair-follicles). At times the affected portions project as much as  $\frac{1}{2}$  inch above the rest and present a real papulous surface, the skin round about being swollen and of well-marked tumid aspect. The older spots finally turn paler, are perfectly smooth and show but slight bran-like deposits.

3. **Tinea Tonsurans in Horses.**—This occurs oftenest on the seat of the saddle, on the croup and flanks, but also upon the head. The several spots range in size from that of a sixpence or shilling to that of a crown-piece and form usually a fairly regular circle. The hair has fallen out or is broken off, and the desquamation of epidermis is considerable, reaching sometimes the stage of slight crusts. The surrounding hair can easily be pulled out, and that which grows again is usually darker in colour.

**4. Tinea Tonsurans in Sheep.**—We find the wool felted, and beneath it are bran-like, scabby parts upon the neck, chest, shoulders and along the back, and considerable itching is manifest. At the outset of the attack small tufts of wool stand out above the rest, and as these increase in number the fleece becomes very ragged in appearance.

**5. Tinea Tonsurans in Poultry.**—This shows itself in loss of feathers, and serious hyperæmia round about the papillæ.

**Diagnosis.**—Ringworm or tinea tonsurans is not very difficult to diagnose. The widely dispersed appearance of the eruption, its round shape, the loss of hair extending outwards from the circumference, the asbestos-like scabby deposits, especially on the heads of dogs (cheeks, lips, eyelids), the slight itch or entire absence of the same, the contagiousness of the disease and, finally, the results of microscopical examination, all help to make diagnosis certain. The evidence of the microscope may be obtained by extracting a few hairs from the edge of a bald place and examining their roots, or by inspection of the scabs, after previously softening them in a 10 per cent. lye of potash and isolating the hair-roots they contain. Viewed by the naked eye, the fungus is characterised by a white, mantle-like envelope on the hair-roots. Confusion with other skin complaints is particularly easy in dogs, in whom ringworm is often hard to distinguish from sarcoptic mange or from the squamous form of acarus eruption without the aid of a microscope. But these two latter complaints are accompanied by intense itching. In cattle, ringworm may sometimes be mistaken for a mercurial eruption, and *vice versa*.

**Prognosis.**—This is so far favourable that cure always follows suitable treatment, if diligently and persistently carried out. In older cattle spontaneous recovery takes place as soon as the scabs have fallen off and the hair, which forms the chief nourishment of the trichophyton tonsurans, has gone with them. We have also noticed a few cases of spontaneous healing in dogs. Only in younger animals can the localisation of the eruption about the mouth cause difficulty in eating and finally death by inanition.

**Therapeutics.**—The prophylactic measures to be first

adopted are, isolation of the affected animals, cleansing and disinfection of their stalls, destruction of all bedding of dogs, and instruction to the attendants respecting the infective nature of the disease. Then, in the next place, the scabs and scales must be thoroughly removed with green soap. Upon the patches thus cleansed the various anti-parasitic remedies may be applied, such as ointment of creolin, carbol, creosote, naphthol or tar (1 : 10), grey, white, and red mercurial ointment (not on cattle), tincture of iodine (1 : 1-5 spirit), salicylic acid with spirits of wine (1 : 10), alcoholic solution of sublimate, or sublimate-salve (1 : 100), solution of arsenic, iodoform ointment; washing with blue-stone, alum, etc. In cases where the affection was not very widespread we obtained good and quick results with the very harmless creolin-salve and tincture of iodine, and also with another safe remedy, viz., alcoholic solution of salicylic acid. We applied them several times daily upon the affected places.

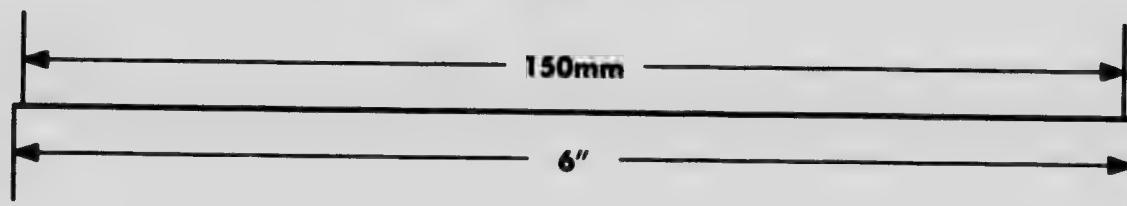
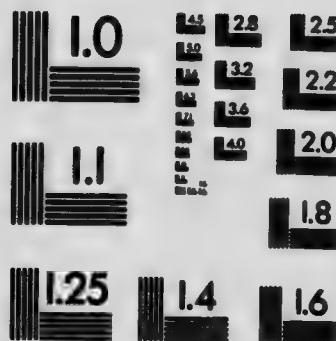
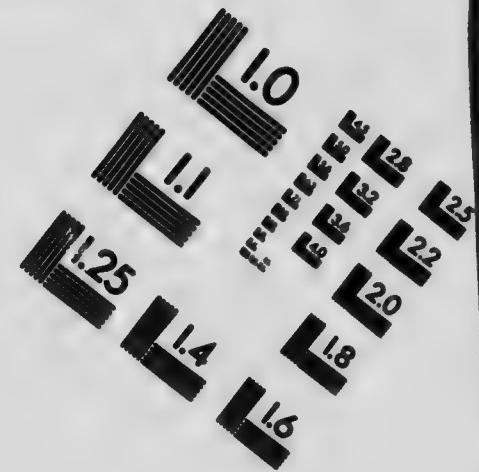
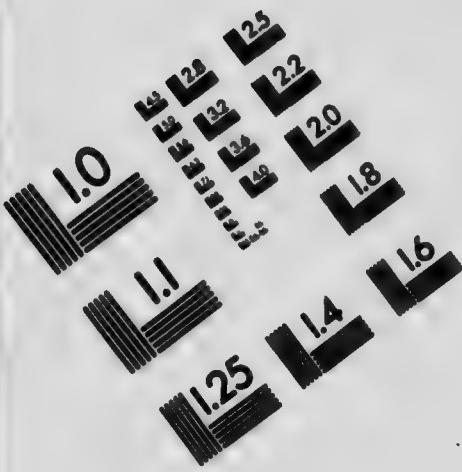
#### FAVUS. SCALL OR HONEY-COMB RINGWORM. TINEA FAVOSA.

**Etiology.**—The favus eruption described as scall or honey-comb mange, and also in poultry as comb-mange, fowl-mange and white-comb (tinea favosa), is caused by a mould-fungus discovered in 1839 by Schönlein and since named after him *achorion Schönleinii*. The disease occurs in dogs and cats, rarely in horses, but also in tame and wild rabbits and in mice. Among poultry the foreign breeds, such as Cochin-Chinas and Brahmases are specially attacked. The fungus may be transmitted to man, and *vice versa* (hence the name "inherited mange"). Cats appear to be mostly infected by eating mice suffering from favus. The favus fungus of man and the larger animals does not seem to be fully identical with that of fowls and mice. The transmission of the poultry favus to horses, cattle or dogs, when attempted by Gerlach, was therefore unsuccessful. In the same way Schütz was only able to transmit its pure culture by inoculation to hens, but not to other animals (rats, guinea-pigs, doves, mice). The fungus lodges most easily upon the tender skin of young animals.

**Morphological Notes on the Achorion Schönleinii.**—The favus fungus forms round discs ("favi") or dish-shaped scabs on the skin, of different thicknesses and sunk in the middle, the so-called scutula ("small shields"),

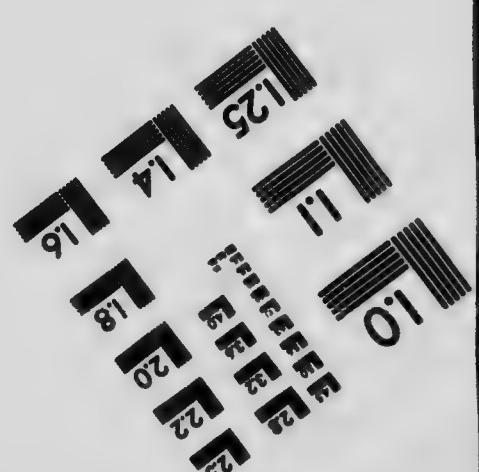
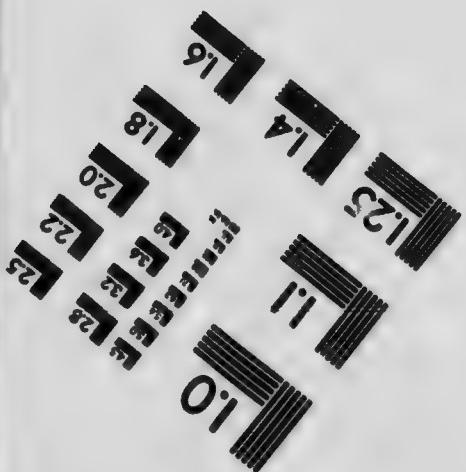


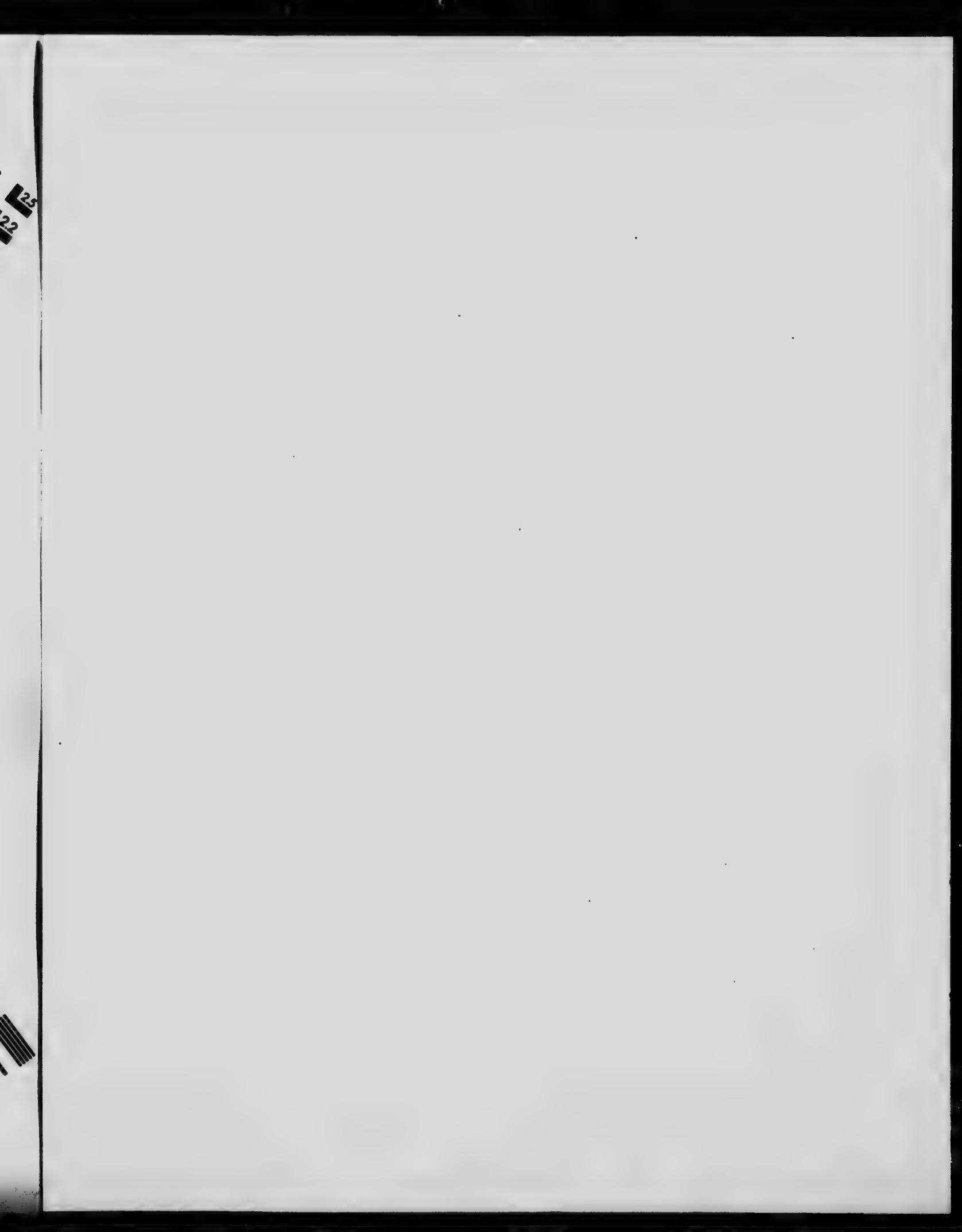
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which consist mainly of fungoid elements and to some extent represent a pure culture of the fungus. Thus we find on inspection of these scutula a thickly felted layer of fungus (mycelium), formed of threads (hyphae) and spores (conidiae). These are morphologically exactly like *trichophyton tonsurans*. The threads are two to five micro-millimètres in diameter, sometimes fine and long-drawn out, without partition, and at others, shorter, thicker, much ramified, segmented, occasionally swollen into club-shape with extremely granulated contents. Between these threads lie a great number of spores, at first cube-shaped, then round or oval and of equal diameter, which have developed from the threads by segmentation. Moreover the favus fungus penetrates into the hair-follicles and feather-papillæ, and finally into the interior of the hair and feathers themselves, which are thereby atrophied at the root and fall out. The threads and spores also luxuriate between the epidermal layers. Schütz obtained a pure culture of *achorion Schönleinii* of fowls upon peptone-gelatine broth at ordinary temperature and transmitted the same successfully to hens.

According to Quincke and Unna, at least three different fungi can produce the aspect of the favus eruption (*favus griseus*, *sulfureus tardus* and *sulfureus celerior*). Others consider these to be merely "freaks" of a fungus which in its morphological and biological behaviour is very variable.

**Symptoms.**—I. *Favus in sucklings*. In these the favus eruption is characterised by dry scabs, which are outwardly greyish-brown to greyish-yellow or silver-grey, and internally range from white to sulphur-yellow, and of a roundish dish-shape, concave in the centre, about the size of a shilling and up to  $\frac{1}{4}$  inch in thickness. These scabs produce atrophy of the hair, with a flat indentation upon the skin. We find them chiefly on the head (ridge of the nose, cheeks, ears, forehead), on the belly, on the outer side of the thighs, and in cats round the claws and in the ear. St. Cyr describes them in dogs and cats as being crusts like mortar, cleft and fissured, of a grey or saffron-yellow colour and tarry consistence, which later scale off and have in the middle a saucer- or basin-like depression. At first they are perforated by a few hairs, which, however, fall off after a time. The skin beneath these crusts is thin and discoloured with blood. In dogs there is considerable itching, but only very little, or none at all, in cats. In horses

the favus scabs may coalesce into long stripes the breadth of a finger. Sometimes animals suffering from favus emit a peculiar smell reminding one of cheese made from cows' milk. The course appears to be in general both benign and rapid. In one case Siedamgrotzky effected a cure after eight days' treatment, and St. Cyr in an average of eight to twelve days, the hair being renewed.

2. *Favus in fowls*, so-called white-comb, fowl-mange (*tinea galli*). The parasitic nature of comb-scab in fowls was discovered almost simultaneously by Leisering, Gerlach and Müller. The following are its phenomena. Small, white spots like mould appear upon the comb and lobes of the ears, which, gradually growing larger, unite to form a white coating over the whole comb. For several months this mouldy coating is restricted to the comb, and changes then into a thick scab. Not until then does the eruption attack the skin of the head, when it very quickly spreads itself over the neck, back, and finally over the whole body. The feathers become erect and fall out, and the whole bird is covered with scaly matter. It grows very thin, acquires a musty, mouldy smell and dies of exhaustion when the process reaches a certain stage.

**Differential Diagnosis.**—The differentiation of favus from *tinea tonsurans* is based mainly upon the presence of the dish-shaped scabs (*scutula*), which also, in contrast to those of *tinea*, are almost exclusively composed of fungus matter, and particularly of hyphae, whereas such accumulations of fungus are not observed in *tinea*; and in the latter there are also fewer hyphae.

**Therapeutics.**—The treatment for favus is the same as that for *tinea tonsurans*. On removal of the scabs, apply the same fungus-destroying agents (creolin, sublimate, carbolic acid, iodine). In fowls spontaneous cure sometimes occurs.

According to Ercolani, the favus fungus vegetates in the horny substance of the hoof, producing hollow places there (so-called seedy-toe) by disturbing the connection between the horny and sensitive layers, and being in this respect analogous to the onychomycosis favosa of man, which penetrates to the interior of the nails. For this reason Ercolani has called this hoof-complaint onychomycosis, and the fungus *achorion keratophagus*.

**CONTAGIOUS ACNE, DERMATITIS CONTAGIOSA PUSTULOSA  
(CANADENSIS).**

*The so-called English or Canadian (American) Horse-pox.*

**Definition.**—This complaint has really nothing in common with the true pox (*variola*) of horses, but represents a peculiar pustular and highly contagious exanthema, with localised lesions, which, in slight cases, are accompanied by formation of simple blisters, pustules and scabs; but in severe ones with more deeply penetrating suppuration and development of papules, ulcers and swelling of the lymph-vessels; but always without any disturbance of general condition, or without itching and benign, although its duration is often prolonged. According to Schindelka, it appears to be identical with the *impetigo contagiosa* of man (Kaposi), which is also an infective pustulous exanthema of the skin of the human head, of similarly benign and typical course.

**Historical Notes.**—English horse-pox, says Axe, to whom it owes its name of *Dermatitis contagiosa pustulosa canadensis*, was first introduced from Canada in 1877. From England it spread, through the export of English horses, over various parts of Europe, where it was observed in different places in 1879. Its identity with *stomatitis pustulosa contagiosa*, which was at first suspected, was later proved to be incorrect. But the exanthema appears to be of older date. In 1841-2, we learn from a description by Goux that an altogether similar, benign, very contagious pustular eruption, which affected chiefly the back, shoulders and croup, attacked the horses of a battery in the French army and affected them all within 14 days, with precisely the same symptoms as the so-called English horse-pox. Hering named the outbreak in question an "infectious tubercular eruption."

**Etiology.**—This epidemic skin affection is mostly transmitted from horse to horse by the rugs, bands, saddles, utensils and cleaning tools used. Consequently, the extent of the exanthema corresponds exactly to the superficial area of a rug, saddle or belly-band. For the same reason the positions occupied by these articles are the favourite seats of the eruption, which seldom passes on to the shoulders or front part of the breast. The head, neck and extremities,

except in rare cases, have remained free. This evidently fixed contagion was declared by Axe to be of mycotic nature. Schindelka regularly found small micrococci in the pus and crusts, which possessed the quality of being easily stained by aniline; and Siedamgrotzky noticed the same in the form of diplococci. The skin affection could be transmitted by inoculation to horses, goats and rabbits; in the latter and in guinea-pigs Siedamgrotzky observed the occurrence in this manner of malignant oedema and septicæmia. Dieckerhoff and Grawitz made a pure culture, in the form of a very short bacillus, of the bacteria of English horse-pox, which they designate *acne contagiosa*. The fungus called "Acne-bacillus" by these two authors forms short rods (half the length of a tubercle-bacillus), whose size is about 0.2 microns, and which divide by segmentation into elliptical or round balls, and which, joining together in twos and fours, form small chains. Both formative elements are very easily stained by watery fuchsin.

**Bacteriological Notes.**—When cultivated, the above-described bacillus forms very characteristic colonies and stale cultures, vegetating luxuriantly on blood serum. Its growth was most rapid at 98° Fahr., but half-an-hour's exposure to a temperature of 176°—194° killed it. When preserved dry, it retained its vitality for four weeks. Strange to say, it produced no putrefaction. It is quite sufficient to rub a few drops of water containing the bacilli upon the normally hairy skin of a horse to produce the typical exanthema. Inoculation succeeded with cattle, sheep, dogs and rabbits. Guinea-pigs were killed within 48 hours by merely rubbing the pure culture of the bacillus upon the sound skin, dying with the symptoms of septic poisoning. In grey mice a subcutaneous injection produced pyæmic phenomena.

**Symptoms.**—After a period of incubation lasting from 6 to 14 days (Schindelka), localised efflorescences appear on the seat of the saddle, on each side of the spine, which differ in character according to the intensity of the disease and the nature of the skin.

i. *In slighter cases* we first perceive round or oval prominences upon the skin, as large as a pea or hazel-nut, which either occur singly or in small groups, covering about the area of a man's palm and not very numerous. The hair above them is ruffled and bushy, the skin sensitive, swollen, and somewhat higher in temperature. These prominences are circumscribed inflammatory areas, and on closer investigation a varying number of isolated and only rarely confluent vesicles and blisters will

be found on their upper surface, which rise to the size of a millet-seed or even a bean, and whose watery contents, at first clear, soon grows purulent (formation of pustules). In one or two days these blisters burst and change to thick, honey-coloured scabs as large as a sixpence or shilling, fastening the adjoining hairs together. About a week later both scabs and hair fall off, and circular spots, without either hair, pigment, scar or desquamation, remain, showing the normal epidermis, so that after about 14 days the entire exanthema has healed, having merely attacked the uppermost skin-layers down to therete Malpighi or papillary body, and without causing any disturbance of general condition. Only a slight swelling of the intermaxillary and of the upper cervical glands is regularly observed, which, however, soon passes away.

2. But to this typical and slight course there are various and frequent exceptions. The causes of these severer cases are more intense infection, the confluence of several pustules and neglect of the eruption at its outbreak in not withdrawing the affected animal at once from work, but allowing the saddle, harness, etc., to increase the irritation and spread the disease. A consequence of such neglect is that the exanthematic process is extended, the inflammatory areas—at first solitary—increase to 50 or 60 in number and the pustules on such areas are multiplied four or five times. New prominences arise near the first ones, and around these again fresh inflammatory areas; the scabs grow very broad and may become larger than a crown-piece. But especially, the inflammation penetrates more deeply into the skin and even into the subcutaneous connective tissue, whereby hard, boil-like and larger nodules are produced, resembling gad-fly boils, which are very painful if touched and possess a suppurating centre, the contents of which may be squeezed out in the form of plugs of pus, leaving smaller or larger ulcerous cavities, which, by confluence, may become as big as a half-crown, and are later filled with granulations. From these larger purulent nodules inflamed lymphatics often radiate in various directions; indeed, suppuration of the neighbouring lymph glands, e.g., the axillary glands, may even take place. This penetrative, purulent infiltration of the skin leaves scars behind it, differing in this from the superficial, pustular lesion. Cure also is greatly delayed, and the process may last, with fresh outbreaks, for five to six weeks, or even two months. But even in these severest forms there is seldom a serious and general illness, and there is no real

itching. In isolated cases the eruption finally settles in the extremities, where it may be complicated with inflammatory swelling of the joints (hocks).

**Differential Diagnosis.**—1. The *true pox* of horses (*variola*) has hitherto only been observed upon the pastern; moreover, this is accompanied with feverish symptoms, which is not the case in Canadian pox.

2. *Farcy* may also show similar phenomena to those of the severer exanthema just described (ulcers, inflammation of the lymph-vessels, contagion). But no blisters or pustules are formed, and there is no such recovery.

3. *Eczema impetiginosum* and *crustosum* and also *acne* closely resemble clinically the contagious pustulous exanthema. But they lack its great contagiousness, neither have such large blisters and pustules been yet observed in eczema. Finally, the latter is marked by much severer itching.

4. Canadian pox can only at first be mistaken for *saddle* or *girth pressure*, but its later course soon removes all possibility of doubt.

**Therapeutics.**—Treatment is unnecessary in the lighter cases, as they heal spontaneously. It is therefore only needful as a prophylactic precaution to isolate the sick animals and disinfect the stable utensils. The horses must also be withdrawn from work. In severer cases treat the open sores with antiseptics. Friedberger reports excellent results from the use of creolin (3 per cent.) and of carbolic acid; Schindelka and Siedamgrotzky of 1 per cent. sublimate solution; Trasböt of 1 to 2 per cent. solutions of blue-stone and sulphate of zinc. These also shortened the duration of the disease.

**Faroy of Cattle** (*Farcin du Bœuf*).—By this name a skin affection is known in France, which resembles the farcy of horses in the worm-shaped form of the swollen lymph-cords, and is produced by a spore-making hyphomycetes (Nocard). This disease seems to have formerly been more common in France, and, according to Couzin, is stationary in the French colony of Guadeloupe. It has also been noted in Germany and Austria. It is a skin disease occurring on the extremities and belly and characterised by corded swellings along the course of the lymph-vessels (lymphangitis) and lymph-glands (lymphadenitis). By formation of metastases in the inner organs (pyæmia), or by general exhaustion, it usually leads to death after a course of years. It may be communicated to cattle and guinea-pigs by inoculation. In the latter, after intra-venous or intra-peritoneal injection, it produces symptoms closely resembling miliary tuberculosis.

III.—SKIN DISEASES CAUSED BY ANIMAL PARASITES.  
MANGE.

A.—*General Description.*

**Definition.**—Under the name of mange (itch, scab, scabies) we understand an infectious skin disease produced by the action of mange-mites; and without such mange-mites there can be no mange. In earlier times the name was used also for non-parasitic skin affections, and before the mite was discovered the true mange was attributed to a dyscrasia of the blood. Among mange-mites in the narrower sense only three kinds are included: the sarcoptes, dermatodectes (psoroptes) and dermatophages (symbiotes), whereas the demodex folliculorum, which produces follicular mange, is usually not reckoned among the itch-mites. Each of these species of mite produces a different morbid condition in the various domestic animals, and each of the latter again possesses its special kinds of mite. The definition of mange must therefore be fixed in every individual case according to the respective species and kind of itch-mite, and we therefore speak more exactly of sarcoptic-mange, dermatodectic-mange and symbiotic-mange. Sheep and dogs are most liable to infection, and horses come next. Cattle, cats, pigs and poultry are more rarely affected. Mange, owing to its highly infectious character, is usually included among the epidemic diseases and should therefore have been treated in this category in our first volume. But on purely practical grounds we have elected to rank it among the skin diseases, as in a text-book of special pathology, the description of clinical results is of the first importance. Moreover, certain forms of mange, such as foot or ear-mange, are of very trifling moment as infective complaints, and possess really only a clinical interest.

**Historical Notes.**—The skin disease known in domestic animals as mange, scab, scall, etc., and in man as itch, boasts a very great antiquity, and was obviously known to Moses as a disease of animals. The mange-mite was also long since discovered. According to Gerlach, it was first mentioned in the year 1174 by the Moorish physician, Avenzoar; and Fürstenberg states that the mites were described under the name of "Suren" in A.D. 1200, in the "Physica" of Saint

Hildegard. On our various domestic animals the mange-mite was discovered as follows :—

- In 1672, on the cat, by Wedel.
- In 1789, on the horse, by Kersting.
- In 1809, on the sheep, by Walz.
- In 1812, on cattle and dogs, by Gohier.
- In 1846, on the pig, by Spinola:

The works of Gerlach and Fürstenberg have been in this department of most conspicuous importance, and among more recent authorities on the mange-mite we would indicate Robin, Mégnin and Zürn.

**Morphology and Biology of the Mange-Mites.**—The mange-mite belongs to the order of acarina (mites), division arachnidæ, family sarcoptes. They are small, round to oval, articulated creatures, usually only visible with a hand-lens or microscope, 0.2–0.8 mm. in size. When fully developed they have four pairs of legs of five segments each, but only three pairs in their youthful stage, such limbs being furnished with suction or prehensile discs, bristles and claws. Their head, thorax and posterior part form one undivided body. From the head project prominent trophi, or feeding instruments, the jaws resembling saws, shears or bristles. As appendices to the outer skin we find scales, hair, spines, bristles, etc. The males are smaller than the females. We should exceed the design of a clinical text-book, were we to enter fully into the details of their structure and manner of life, and must, therefore, refer students to the special works on general pathology and the science of parasites. The females lay from 20 to 24 eggs at once, which hatch out in four to seven days into larvæ, which, after three or four changes of skin, are capable of reproduction in fourteen to seventeen days. Gerlach has calculated that in this manner about one and a half millions of sheep-mites may spring from a single female in 90 days. The females die three to five weeks after laying their eggs, the males live to be about six weeks old. The mites can exist isolated in moist air or on moist dung, etc., for a period of six to eight weeks, and in dry air two to three weeks. The eggs will keep for two to four weeks on a moist surface, but on a dry one only four to six days. Both mites and eggs are killed in about an hour by a temperature of 104° to 140° Fahr.; but a moderate heat stimulates them, so that they become

more active and propagate more rapidly in warm stables, under rugs and in summer time.

According to Gerlach, the mange-mites are most speedily killed by the following drugs:

By creosote in  $\frac{1}{2}$  to  $\frac{1}{4}$  minute; caustic potash (1:24), in 2 minutes; sulphurated potash (1:10) in  $\frac{1}{2}$  to  $\frac{1}{4}$  hour; oil of turpentine, in 5 to 9 minutes; tar, in 8 to 18 minutes; petroleum, in 7 minutes; tobacco (1:5), in 10 to 20 minutes; tobacco (1:10), in 2 to 5 hours; tobacco (1:50), in 4 to 10 hours; green soap, in  $\frac{1}{2}$  to 1 hour.

According to the more recent tests of Müller, the several mange remedies stand, as regards their strength and rapidity of action upon the various kinds of mange-mites (and acarinae), as indicated in the following table:—

Name of Drug.	Acarus.	Sarcopis.	Dermatophytes.
<b>After being treated with :—</b>			
Cresote 10%	at once. in 6 minutes at most. in 5 minutes at most. at once. in 15 minutes. not after two hours.	at once. in 6 minutes at most. almost at once. in 18 minutes at most. in 45 minutes at most. not after 2 hours.	almost immediately. at once. in 15 minutes at most. in 40 minutes at most. not after 2 hours.
Carbolic acid 5%	in 15 minutes.	in several minutes.	in 45 minutes at most. not after 2 hours.
Sublimate 2%	in 15 minutes.	in a few minutes.	in 45 minutes at most. not after 2 hours.
Sublimate 1%	in 15 minutes.	in a few minutes.	in 45 minutes at most. not after 2 hours.
Sublimate 0.6%	in 15 minutes.	in a few minutes.	in 45 minutes at most. not after 2 hours.
Oxy-naphthoic acid 5% (spirit)	in 25 minutes at most. in about 30 minutes.	in about 13 minutes.	in 45 minutes at most. not after 2 hours.
Salicylic acid 10%	in 24 minutes. not after 2 hours.	in 95 minutes.	in 45 minutes at most. not after 2 hours.
Tetethyl	in 85 minutes.	not after 2 hours.	in 20-25 minutes.
Sulphurated potash 20%	at once.	not after 2 hours.	almost at once.
Sesqui-jodol-mercury 2.5%	in 24 minutes.	in 5-6 minutes.	in 5-6 minutes.
Arsenic 1%	not after 2 hours.	at once.	at once.
Decotion of tobacco 15%	in 85 minutes.	in a few minutes.	almost at once.
Wood-tar	at once.	in 6 minutes.	almost at once.
Tar-liniment	at once.	in 20 minutes.	in 20 minutes.
Peruvian balsam	in a few minutes.	in 25 minutes.	in 25 minutes.
Stinking animal oil	in 6 minutes.	in 95 minutes.	in 95 minutes.
Naphthalene oil 10%	in 20 minutes.	in 2 hours.	in 2 hours.
Carbolic oil 10%	in 25 minutes.	not after 2 hours.	not after 2 hours.
Petroleum	in 25 minutes.	not after 2 hours.	not after 2 hours.
Naphthalene oil 10%	in 95 minutes.	not after 2 hours.	not after 2 hours.
Oxy-naphthoic acid oil 10%	in 2 hours.	not after 2 hours.	not after 2 hours.
Sesqui-jodol-mercury oil 20%	in a few minutes.	in 15-18 minutes.	in about 10 minutes.
Mercurial salicylate oil 20%	in a few minutes.	in 15-18 minutes.	in 15-18 minutes.
Salicylic oil 5%	in a few minutes.	in 15-18 minutes.	in 15-18 minutes.

From the foregoing it is plain that creosote, creolin, tar, animal-oil and Peruvian balsam act immediately and with almost equal strength upon *acarus*, *sarcoptes* and *dermatoryktes*. Carbolic acid, naphthol, petroleum, tobacco and sulphurated potash considerably more slowly, and are more fatal to *sarcoptes* and *dermatoryktes* than to *demodex*. But, on the other hand, sublimate and soziiodol-mercury poison the latter more quickly. According to Kippel, iodine in the form of Lugol's solution (1 : 80) kills *dermatodectes* in ten minutes, *sarcoptes minor* in six; and 1 : 40 kills *demodex folliculorum* in five minutes:

**Species and Kinds of Mange-Mites.**—The mange-mites as above described are divided into three species.

1. *Burrowing-mites, sarcoptes*:

2. *Sucking mites, dermatocoptes* (Fürstenberg); by Gerlach termed *dermatodectes*, and by Mégnin *psoroptes*.

3. *Scale-eating mites, dermatophagus* (Fürstenberg); by Gerlach called *symbiotes*, and by Mégnin *choriopetes*.

A. **The Sarcoptic-mites** bore themselves burrows under the upper skin and feed upon the cells of the rete Malpighi, which contain plasma. They also propagate in these burrows, producing an intense inflammation of the skin. They are characterised by their tortoise-like shape and extraordinary smallness (0.2—0.5 mm.), so that they can only be recognised by the microscope. Their heads are horse-shoe shaped and have two strongly-developed, conical, shearing jaws; the feet are short and stumpy, provided with pedunculated, tulip-shaped, prehensile discs, which in the male are attached to the first, second and fourth pair of feet, and in the female to the first and second only. As regards the sub-division of *sarcoptes* into varieties opinions differ. Fürstenberg has been guided therein more by morphological considerations, and Gerlach by biological. But attention has often been called to the great inconstancy of these varieties and to their different and remarkable deviations from type (Zurn, Johne). We have adhered in the following to the sub-division made by Siedamgrotzky, in which there are two chief kinds: (a) the large burrowing-mite and (b) the small burrowing-mite.

(a) To the large burrowing-mites belong: 1st, *Sarcoptes scabiei* of man, the horse, sheep, lion and tame rabbit; and 2nd, *sarcoptes squamiferus* of dogs, pigs, sheep and goats.

(b) The small burrowing-mites, *sarcoptes minor*, occur in cats and in tame rabbits.

B. **The Dermatodectic Mites** live on the outer surface of the skin and, by means of their mouth-apparatus or trophi, suck blood, serum and lymph from it, clinging the while to scales and hair. They also cause inflammation of the skin by pricking it. They are characterised by their size (0.5—0.8 mm.), so that on a dark background they may be recognised with the naked eye, and certainly with a magnifying glass, being identified by their long feet, long, pointed head, long, straight, piercing jaws, and lastly by their tulip or trumpet-shaped suction-discs, which are attached to articulated peduncles, occurring in males upon all

four feet and in females upon the first, second and fourth pair of feet. According to Zürn, the dermatodectes are divided into :

- (a) *Dermatodectes communis*, on horses, cattle and sheep.
- (b) *Dermatodectes cuniculi*, the ear-sucking mites of the rabbit.

Even among the sub-varieties of dermatodectes there occur not unimportant diversities. Thus Zürn has described such an one on horses, and we ourselves have often noticed slight variations of structure.

C. The **Symbiotic Mites** inhabit especially the upper skin-surfaces of the extremities, feeding upon the younger epidermis scales, which they gnaw away. They are also moderately large (0.3—0.5 mm.) ; their outlines are, therefore, visible to the naked eye or with a magnifying lens. They are characterised by short, stumpy, conical heads, which are broader than long, an oval body slightly notched on its edges, long feet, tumbler-shaped suction-discs, which in males are found on all four feet, and on females on the first, second and fourth pairs. The male is also furnished on the hinder part of the body with two prehensile organs, and the female with two cylindrical cones. Zürn thus sub-divides them :

- (a) *Symbiotes communis* :
  - (1.) *Symbiotes equi* and *bovis*, which produce foot-mange and rump-mange.
  - (2.) *Symbiotes ovis*, which causes the foot-mange of sheep.
- (b) *Symbiotes felis*, *canis* and *cuniculi*, producing ear-mange.

**General Character of the Various Kinds of Mange and Classification of the Acarina as found on Various Domestic Animals.**—The three varieties of mange-mites produce clinically different kinds of mange, which are due to the manner of life of the acarus.

1. *Sarcoptic-mange* begins by preference upon the head and those parts of the body which are least protected by hair, upon which, on sheep for instance, it exclusively occurs. But it may spread from the head to the neck, and finally over the whole body, as it does in horses, dogs and swine. On the dog it prefers the ears, the vicinity of the elbows, the lower breast, belly and inner surfaces of the extremities.

2. *Dermatodectic- or psoroptic-mange* prefers the sheltered parts of the body, thus in horses the intermaxillary space, the inner side of the thighs, the neighbourhood of the sheath, the roots of the mane and tail ; but may spread over the entire body when proper attention is not given to the skin. With sheep the dermatodectes mites seek first those parts which are protected by wool, and may therefore attack the whole body except the head.

3. *Symbiotic-mange* attacks chiefly certain parts of the body, thus in horses (and sheep) the lower portion of the feet, so-called foot-mange ; in cattle around the root of the tail,

so-called rump-mange; in dogs, rabbits and cats, the external ear-passages, so called ear-mange.

Upon the several domestic animals the following mites occur:—

A. On **Horses**: Sarcoptes scabiæ, dermatodectes communis, symbiotes equi; *principal-mange*: *Sarcoptic-mange*.

B. On **Cattle**: Dermatodectes communis, and symbiotes bovis; *principal-mange*: *Dermatodectic mange*.

C. On **Sheep**: Sarcoptes squamiferus, dermatodectes communis, symbiotes ovis; *principal-mange*: *Dermatodectic mange*.

D. On **Goats**: Sarcoptes squamiferus, dermatodectes and symbiotes; *principal-mange*: *Sarcoptic-mange*.

E. On **Dogs**: Sarcoptes squamiferus and symbiotes canis; *principal-mange*: *Sarcoptic-mange*.

F. On **Cats**: Sarcoptes minor and symbiotes felis; *principal-mange*: *Sarcoptic-mange*.

G. On **Pigs**: Sarcoptes squamiferus.

H. On **Rabbits**: Sarcoptes minor, dermatodectes and symbiotes cuniculi; *principal-mange*: *Sarcoptic-mange*.

#### **Transmissibility in General of Mange to Animals and Man.—**

The following is what is at present known upon this point:

1. All the kinds of sarcoptes of animals may be transmitted to man and exist on him permanently. On the other hand, dermatodectes and symbiotes quickly die away on the human skin, or only produce very slight signs of inflammation there.

2. From sheep, pigs, dogs and cats sarcoptes are transmissible to the horse.

3. The sarcoptes of horses, sheep, goats and cats are transmissible to cattle.

4. The sarcoptes of goats may be transmitted to sheep.

5. The sarcoptes of man, the pig, cat, sheep and goat are transmissible to the dog.

6. The sarcoptes of the goat may infect pigs.

7. The sarcoptes of man may also infect rabbits. From the foregoing may be gathered that hitherto only the transmission o' the various kinds of sarcoptes from one animal to another has been observed.

**General Phenomena of Mange.—**The disease develops only after previous infection. The period of incubation after

transmission of the mites, that is, the time which elapses before any change is noticed upon the skin, varies according to the number of mites so transmitted. If they be only few, then four to six weeks may elapse before there is any manifest outbreak of disease; on the other hand this may occur within fourteen days. The infection is caused either by direct transmission (straws, covers, utensils, brushes, etc., or attendants). Poorly tended animals and such as are feeble and suffering from much scurf on the skin are peculiarly predisposed to infection by mange parasites.

The first symptoms provoked by the bites of the mites are a dotted redness (so-called stigma), nodules and blisters, an increased proliferation of epidermis and a deposit of crusts and scabs. From the very first these skin changes announce their presence by an intense itching, which, particularly in the night, under the rays of the sun or after sharp exertion, may become exceedingly acute. The animals then rub, bite and scratch the affected spots, and the mechanical irritation thus wrought very considerably alters the otherwise simple phenomena of the eruption. The hair falls out or breaks off, the wool grows shaggy, the skin discoloured with blood, and inflammatory processes are set up, which finally become chronic and cause permanent changes. Thus, at the climax of the disease we find the skin partly covered with impetiginous scabs and partly weeping and bloody, even ulcerously degenerated, thickened and creased.

**Course.**—This depends on various circumstances. First the kind of mite must be considered; whereas symbiotes produces mainly a localised mange, the eruption in sarcoptic-mange spreads readily over the whole body, though this occurs also with dermatodectes. Then the nature of the skin, the degree of attention paid thereto, the quarters occupied by the animals, the season of the year, etc., are all of importance. In summer the greater heat causes a more obstinate course than in winter. Well-tended horses, such as military horses, will show only a slight degree of eruption for a long time. The ailment always decreases in sheep after shearing, because the mites are deprived of the sheltering wool. Finally, the intensity of the process may be greatly influenced by previous washing of the skin with suitable remedies. In general the course of mange is chronic. Spontaneous cures have not yet been observed; for the intermittent cases, in which the eruption

considerably abates at certain seasons, cannot well be regarded as cures. When once the mange has spread over the whole body, general symptoms usually also appear. In consequence of the protracted irritation and loss of fluid, the animals go back in condition and growth, and may even perish of exhaustion and cachexia.

**Diagnosis of Mange in General.**—The recognition of mange as such can be based upon certain peculiarities of the eruption, apart from the presence of the mites. Among these are, first of all, its epidemic appearance and great infectiousness, the localised development of the eruption at the outset followed by very rapid spread. Pruritus is also much more marked than with other dermatoses. The locality affected by the mange likewise affords points for guidance. Thus, an eruption confined to the head, or spreading thence, with the above-named characteristics, indicates sarcoptic-mange; one restricted to the limbs or neighbourhood of the anus points to symbiotic mange; and, finally, one which seeks especially sheltered parts is likely to be dermatodectic-mange. But the surest diagnostic method is identification of the mite. This is best done by the microscope, when the scabs and crusts with their deeper layers must be taken off, cleared with a 10 per cent. solution of liquor potassæ, and then examined under a glass of low power. In the sarcoptic-mange of dogs the portion so taken off must go very deep, so that one must either scrape the skin with a sharp knife until it bleeds, or cut off a thin layer of the epidermis with a razor or scissors. Formerly it was recommended to bind a mange-scab on the human arm, when itching and slight inflammation would arise from sarcoptic-mange within twelve hours, and from dermatodectic within two hours. But the microscopical test is much surer. The two larger mites—dermatodectes and symbiotes—may be rendered visible to the naked eye, or to a magnifying glass, by laying the scabs on black paper or a sheet of glass covered with a watch-glass and letting them be warmed in the sun-light, as under the influence of the warmth the mites move actively about or adhere to the watch-glass. To distinguish the various manges, see the earlier paragraph on the several forms of mange in different domestic animals.

**Prognosis of Mange in general.**—An estimate of the result of mange depends first of all upon the species of the

mite and the extent and age of the disease. A sarcoptic-mange spread over the whole body, for example, is very difficult to cure in a horse. This difficulty has not, in our opinion, been sufficiently appreciated. On the other hand, symbiotic mange in horses is very easily curable. Wide-spread dermatodectic mange of long standing is also very hard to remove from sheep, while in horses it may be easily combatted if of limited extent. Sarcoptic-mange in cats frequently ends in the death of the affected animal. Wide-spread sarcoptic and dermatodectic manges are therefore prognostically the most unfavourable; The same manges are more favourable when only local, and most so is symbiotic mange. The condition of the animal as regards nourishment is also an important factor in prognosis. Badly nourished or very young animals are hard to cure, being less able to endure the somewhat severe treatment. Weather and season also influence prognosis, in so far as with sheep, for instance, baths can only be adopted in warm weather without injury. Further, the character of the hairy covering and the individual susceptibility to certain medicinal remedies must both be taken into account. There is much less chance of good results in treating thick-haired animals than with thinly-haired or shorn skins, and in sheep such treatment is usually quite fruitless until they are shorn. Many animals also are much more sensitive to certain drugs or forms of manipulation than others. Thus, carbolic acid cannot be used on cats without risk of poisoning. Cats, goats and poultry are moreover very sensitive to baths. Finally, the position upon the body of the mange is also an important consideration, as some parts are unsuited for the application of strong drugs, such as the neighbourhood of the eyes. The possibility also of fresh infection through unfavourable external conditions must not be overlooked.

**General Remarks on the Treatment of Mange.—**  
Mange can only be cured by the use of substances which destroy the mites, of which many are in use. The mildest and also the weakest are the alkalis : potash-lye, green soap, the carbonates of soda and potash, also burnt lime, chlorinated lime, sulphate of potash, ammonia and the ammoniacal urine of cattle. As all these remedies also soften the scabs and epidermal-scales, they are mostly used along with stronger ones, to help the latter to penetrate the skin. The various sulphur preparations also act very mildly, and in combination with the alkalis

generate sulphuretted hydrogen, which has a very fatal effect upon the mites. Still more intense is the action of tobacco, either as a lye or a decoction; also of arsenic and mercury, the latter in the form of sublimate or the less severe grey mercurial ointment. But most efficacious are the products of destructive distillation of vegetable and animal substances, above all creolin, lysol, carbolic acid, creosote, tar, benzol and stinking animal-oil. With regard to the form and combination of these in individual cases see a little later on. Here we will only raise the following general points concerning the treatment of mange.

1. As a prophylactic precaution mangy animals must be *secluded* from sound ones as much as possible, and care be also taken that during treatment they are not afresh infected by other animals or by agents of transmission (covers, cleaning tools, straw, stalls). All these, and the entire stable, should be carefully disinfected.

2. Before applying medicaments let the skin be thoroughly cleansed from all scales and scabs.

3. In using drugs for the destruction of the mites have regard to the *constitution and species* of the sick animal, and to any *pre-existing wounds* upon its skin. Feeble animals require gentler treatment, and there are drugs which may not be used on certain kinds of animal (mercury for cattle, or carbolic acid for cats). Sheep should not be rubbed or bathed with intensely poisonous mange-remedies immediately after shearing, because the skin then possesses greater power of absorption, owing to the numerous small wounds on the same.

4. Those *places known to be affected* by the disease must be most *vigorously treated*; but treatment must extend, as a rule, to their non-infected surroundings, and in sheep should always cover the whole body, as every part is liable to infection.

5. The *application* of medicaments must be *repeated* after a certain interval, as most remedies can only destroy the mites, but not their eggs, and from these latter fresh mites develop. In some circumstances, especially where the mange is very widespread and inveterate, a *third* application is advisable to kill any mites which may hatch out after the second. But for the more deeply penetrative sarcoptic-mites even a third dosing with mange remedies does not suffice, but rather a repeated and even prolonged treatment is necessary in order to ensure a radical cure, especially with a sarcoptic-mange of long standing in horses.

## B.—MANGE IN THE SEVERAL DOMESTIC MAMMALIA.

*a. Mange in Horses.*

**i. Sarcoptic-Mange.**—This generally breaks out on the head, neck and shoulders and on the walls of the breast; but also, according to the mode of infection, may start from the withers, seat of saddle or belly-band, or on the hind legs and flanks. It begins with small, circumscribed bald patches, which gradually enlarge and may attain considerable dimensions. But it is only rarely that the eruption covers all the body, but rather keeps to the head, neck and shoulders. Its chief *symptoms* are an extraordinary itching, which is most intense at night, in hot sunshine or after heating of the body, and excites the animal to much rubbing, etc. If the attendant scratches the affected place it will evince lively signs of pleasure. The changes effected on the skin consist in small nodules, from which the hairs fall off, or are held together by a sticky fluid. Occasionally vesicles also are formed. As the disease proceeds, a very active desquamation of epidermis takes place on the nodules, with a gradual increase of scab-formation. Thus we have seen mange scabs nearly  $\frac{1}{2}$  inch thick, which consisted mainly of epidermal-cells, and in which the burrows of the mites could be traced in storeys. The skin also grows thicker and parchment like in sympathy with this inflammatory process, is crumpled into swollen folds, becomes cracked and fissured, discharging matter and pus, and has in places a sore and bruised appearance. In prolonged cases, and when the disease spreads over the whole body, the poor brutes have a most wretched aspect, quickly grow thin and may even die cachectically. The eruption is infectious for man, and attacks especially the hands and arms of the surgeon and attendants. In 1791, 200 hussars of one regiment were infected by their horses (Sik).

According to official statistics, horse-mange is very rare in Germany as compared with sheep-mange, only about 550 cases annually being reported, chiefly in the provinces on the Russian frontier. In Austria it is much commoner—average 1,255 per annum, of which 13.3 per cent. proved fatal.

The *diagnosis* of sarcoptic-mange is difficult to establish at first, because the mites are then not easily discovered. Only rarely does one succeed in finding a single mite or its case, and at this stage the most careful search is often quite fruitless.

The only important diagnostic symptom is therefore the extraordinary itching, which in the non-parasitic eczema (so-called nodular scabies or summer-mange) is by no means so intense. This sensation, combined with the slight skin-changes, led formerly to this being regarded as prurigo or pruritus, skin affections which, however, evidently corresponded in their chief symptoms to the initial stages of mange. In very long-standing cases of mange which have been much neglected, the mites are easily found.

[The *prognosis* of sarcoptic-mange in slighter cases is favourable, but quite the contrary in severe ones. As regards its curability, sarcoptic-mange differs greatly from dermatodectic mange, being very much more difficult to cure and requiring in its older and severer forms a much more vigorous treatment, lasting often for months. Relapse is, moreover, very frequent, and though this is usually regarded as the result of a fresh infection, yet it often occurs when all such possibility has been sedulously avoided.

*Treatment* consists first in cleansing the skin, softening the scabs with 5 to 10 per cent. carbolic or creolin-glycerine, and application of soft soap to facilitate their removal. The soap should be left on for about twenty-four hours, and, if necessary, the hair clipped off close. Having thus prepared the skin, apply the mange remedies in the form of ointment, liniment and washes. In slighter cases we commend Vienna tar-liniment, which is made up as follows: Tar and sulphur, of each 1 part; green soap and spirit, of each two parts. Rub well into the skin with brushes, leave it on for 6 to 8 days, renewing it wherever rubbed off, and then wash all clean, repeating the whole process twice or thrice. In most slight cases the result is permanent. When the mange is inveterate and widespread, it is well to alternate with other remedies, but only too often they all prove unavailing. Besides Vienna tar-liniment, we may recommend creolin liniment, prepared from creolin and green soap, of each 1 part and spirit 8 parts. In addition one may use carbolic acid or creosote (1 : 10 oil or spirits), arsenic-vinegar, sulphur ointment, styrax, solutions of creolin (3 per cent.), ditto of sublimate (1 to 3 per cent.), as also decoctions of tobacco (5 per cent.). But washing with sublimate alone does not seem very effectual, and we only obtained permanent cure after very many applications of a 2 to 3 per cent. solution, or after an addition thereto of a 15 per cent. tobacco decoction;

**2. Dermatodectic Mange.**—This begins with sharply-defined spots, somewhat larger even from the first, and mainly situated on the sheltered parts of the body, such as the roots of the mane and tail, in the maxillary space, on the sheath, on the udder and inner sides of the thighs. We have also seen it in other places, such as the withers, back and croup. The earliest phenomena are nodules, which represent an inflammatory reaction of the skin against the injuries caused by the bites of the mites, and from which a lymph-like fluid oozes, which dries into a scab. Increased desquamation of epidermis also takes place, with falling-out of the hair. The deposits of scales and scabs may in course of time become as thick as the finger. Later, the entire skin gradually thickens, takes on a leathery appearance, and crumples into folds. The itching sensation is here also somewhat considerable, and serious inflammatory conditions of the skin may result from rubbing, which may lead to penetrative suppuration and bloody sores.

The differentiation of dermatodectic mange from the sarcoptic form is based—in addition to identification of the mite—upon its preference for sheltered parts of the body, the formation of clearly-defined areas, and its slower spread. But we must admit that, in cases spread over wider areas, its clinical aspect very much resembles that of sarcoptic-mange. Any confusion with *eczema squamosum* (*scaly scabies*) and mane-mange may be avoided by observing the previous acute itching of dermatodectic mange and the sharply-defined outline of its areas—this apart from microscopical investigation.

*Recovery* is in cases of dermatodectic mange usually much more rapid than in the sarcoptic variety, and with the relatively milder remedies used one sooner obtains the desired result. *Treatment* with creolin or carbolic soap, or with creolin or carbolic glycerine, in the proportion of 1:10, generally suffices; or Vienna tar-liniment may be applied. The latter is, however, less suitable when the scabs are very thick. On an average even severer cases may be cured in from fourteen days to three weeks. Only with stallions, which show a formation of deep folds on the upper margin of the neck, does a cure appear to be difficult, or even impossible (Trasbôt).

**3. Symbiotic-Mange.**—This variety, which has also been called foot-mange, is characterised by a great preference for the feet. It occurs oftenest in the pastern, whence it spreads to the fetlock and to the surfaces of the hind and fore shins,

and may even rise to the hock, knees and elbows. In rarer cases it ascends to the shoulders, neck and lower and upper thighs. But its development is remarkably slow, so that a distinct extension is only perceptible after a lapse of months. Its *symptoms* are first seen in a repeated and, one might say, spasmodic stamping (this is often the earliest sign) and even kicking, especially at night, also in rubbing and gnawing of the fetlocks. If we examine these, we find abundant desquamation of epidermis and loss of hair, followed later by the appearance of scabs, cracks and fissures; the skin becomes brittle and rough, thickens and, in prolonged course, reveals horny, papillary proliferations. The *diagnosis* of this foot-mange is guided by its position, the desquamation of epidermis and the violent itching. This latter item distinguishes it also from an ordinary eczematous greasy heels. It is only when it, quite exceptionally, spreads over the whole body that it can be mistaken for sarcoptic-mange. *Treatment* consists in the application of creolin or carbolic-glycerine, carbolic soap or tar-ointment (1 : 10). These remedies suffice to effect a cure in a short time.

Mégnin describes symbiotic mange as "intermittent mange." He maintains that it appears in winter and vanishes in spring, but that during this period the mites remain alive, but live during the warmer season upon the more abundant hair secretions, whereas in winter they are compelled to attack the skin itself. According to Schwarz, the frequent wounds upon the pasterns are caused by the horses' own restlessness when suffering from foot-mange, as a result of which their pasterns often become entangled in the halter-chain.

#### SCAB IN SHEEP.

**Statistical Notes.**—All three kinds of mite infest sheep, but dermatodectic mange is by far the most important form of the disease. How serious its ravages sometimes are may be shown by the following figures. In 1884 an official report showed that in Germany nearly a quarter of a million sheep suffered from scab, and in Alsace-Lorraine the proportion affected reached nearly 61 per cent. of their entire sheep. In later years there has been a considerable diminution in these figures, but the loss to Germany in wool alone must amount to many hundreds of thousands of pounds.

**Pathogenesis.**—The mode of transmission of the mange-mite is usually direct; an infection by some carrying agent being less frequent. Transmission occurs most easily when shorn

sheep suffering from scab are turned out among a flock still unclipped. It takes place also more freely in warm temperatures, in summer, in the stable, or when the animals lie together, as the mites are then much livelier.

**Phenomena of Dermatodectic Mange.**—The favourite spots for the dermatodectes mites are those covered with wool, while the more naked parts, and especially the head, are neglected. By far the oftenest they attack the flanks and root of the tail, spreading thence over the back, sides, neck, shoulders, etc. The eruption always develops from small spots which, according to the mode of infection, are either solitary or spread in varying numbers over the body. The progress of the development is as follows: On dividing the woolly coat we see at first flat nodules the size of a millet-seed, of pale yellow or reddish colour, which have been caused by the bite of the mite, and, according to the number of these, are either found singly or united into larger, flat prominences as large sometimes as a half-crown. The skin round about is reddened, and the whole region at first painful when touched. Blisters soon appear on the top of these nodules and small pustules, which burst and dry up. At the same time active desquamation of epidermis sets in, which, in combination with the more abundantly secreted fatty sweat, forms a yellowish deposit, and when conjoined to the exuded matter of the blisters and pustules, produces often a very thick, hard, yellowish-brown mange-scab, beneath which the parasites flourish. Upon shorn skins this scab assumes an aspect resembling parchment. The wool is loosened at the root and glued together by the fluids discharged from the nodules, and as the wool grows the mange-scab is also lifted up with it. The entire fleece gradually becomes shaggy and flaky, larger or smaller tufts rise above the general surface, which finally fall or are rubbed off. The separate wool-fibres are lustreless, pale in colour and easily pulled out. The skin below the scabs has lost its lustre and smoothness, is rough, dry, crinkled, thickened and covered with fissures and chinks, and, in consequence of much rubbing, is ulcerated.

Along with these local changes of the skin there is also very intense itching; indeed, this is usually the first symptom of sheep-scab. The animals rub, scratch, gnaw, pinch and pluck at the affected spots, shake and roll themselves about, sink their backs, and if anyone scratch them, evince the liveliest pleasure, quivering their lips, scraping involuntarily with the hind feet,

shaking their heads, clashing their teeth, wagging the tail violently and pressing up against the hand which rubs them. This itching is most severe in warmth and towards evening. The constant rubbing hastens the fall of the wool and causes more or less serious inflammatory and ulcerous affections on the skin. If the mange spread far, the animals lose flesh, owing to the persistent irritation, loss of fluids and hindrance in feeding and rumination. Feeble animals consequently often perish (hydræmia, general cachexia). The course of the disease always shows considerable aggravation in autumn and winter, being very appreciably checked by shearing and washing. Finally, the tenderer skins of the finer breeds seem to suffer more intensely than those of ordinary sheep.

**Diagnosis.**—For the identification of scab the following points are of importance. 1. *Proof of infection*, or of the extended presence of the disease in a flock. 2. *The characteristic itching* and restlessness on the part of several animals when inspecting the flock. We must, however, point out here that if an animal be vigorously rubbed by anyone examining it, no itching may be observed, but only signs of pain, a fact often noticed at the commencement of an outbreak of scab eruption. Also that the shepherds know how to suppress all expressions of relief and pleasure by firmly holding the sheep during its examination by the surgeon, and can also temporarily remove the itching sensation by the use of certain remedies. 3. *The condition of the wool*; a flaky, shaggy fleece, easy extraction of the wool and the presence of bald patches are suspicious signs. 4. *Careful examination of the skin*, especially about the loins, root of tail, back and shoulders. These parts should be thoroughly palpated with the finger-tips, and the skin exposed by parting the wool. Any discoloration of the wool by remedies previously applied by the shepherds, so-called grease spots, must be carefully noted. 5. But diagnosis can only be rendered positively sure by *microscopical proof of the presence of the mites*.

**Differential Diagnosis.**—The following may be confounded with dermatodectic mange:—

1. So-called *rain-rot*, an eczema produced upon the skin by persistent rain and which, like scab, may spread through a whole flock. But the rapid healing of this eczema on return of dry weather or removal to cover, the absence of itching, combined with the method of its origin, all facilitate differentiation.

2. *Other parasitic skin eruptions*, especially those caused by lice. In such cases a careful examination of wool and skin may prevent confusion.

3. *Excessive accumulations of wool-fat*, by which, under some circumstances, the hairs are glued together. Apart from the absence of itching, the skin will be found in such cases to be perfectly clean, smooth and lustrous.

4. *Wilful erosions of the skin*, by caustic remedies, may be recognised partly by the colour of the substance used, and partly by the intense uniform slough, though sometimes it may not be easy to distinguish them from mange-scabs. But the later course and microscopical investigation will furnish sufficient data for a sound opinion.

**Therapeutics.**—In the treatment of sheep-scab a distinction is drawn between healing by inunction and by dipping. The first is only a provisional operation, intended as a preparation for the latter, or performed, for instance, in winter, because the other cannot then be undertaken. It consists in rubbing of local scab areas with mite-destroying ointments or liniments (carbolic soap, carbolic oil, creolin-liniment, grey mercurial ointment, tobacco-juice, petroleum). This rubbing treatment does not suffice for a radical cure, for the simple reason that only the visibly-affected spots are anointed, while smaller centres spread perhaps over the whole body, remain untouched. Objection has been raised from many sides to the use of grey mercurial ointment on the ground of its poisonous action, which may even extend to the lambs of the ewes thus treated. It is wiser, therefore, to replace it by other and safer remedies. For dipping purposes numerous sheep-dips have been introduced, on the value of which divers opinions are held. The following are the chief of these :—

I. The so-called **Walz's dip**, the oldest known bath for mange, introduced by the discoverer of the *dermatodectes* mite. It consists of 4 parts of quick-lime, 5 parts of potash (or 60 of wood-ashes), 6 parts of stinking animal-oil, 3 parts of tar, 200 parts of ox urine and 800 parts of water. This is certainly the cheapest of all sheep-dips, but is by many considered too weak, as severe cases or those of long standing cannot be healed by it; moreover, it dyes the wool brown. It is therefore only appropriately applicable as a preparation for one of the other stronger mixtures.

2. **Gerlach's Scab-dip** consists first of a preparatory bath of 2 parts of potash, 1 part of quick-lime and 50 parts of water ; and the mange-bath proper of a 3 per cent. tobacco decoction. According to Roloff, this latter is better in a 5 per cent. strength. This dip of Gerlach's has generally been recognised as very effective, especially since Roloff's modification. It is, however, very troublesome to prepare, and the preliminary bath affects the hands injuriously.

3. **Zündel's Scab-dip** consists of  $3\frac{1}{2}$  lbs. of commercial carbolic acid,  $2\frac{1}{2}$  lbs. of quick-lime,  $6\frac{1}{2}$  lbs. each of soda and soft soap mixed in 57 gallons of water, this quantity sufficing for 100 sheep. This very cheap dip (costing about 2s.) may be improved, according to Kayser, Ostertag and others, by using a 2 per cent. decoction of tobacco instead of water (about 11 lbs. of tobacco to 57 gallons of water), whereby, it is true, the cost is trebled, but the effect greatly augmented. This dip is then a combination of those of Gerlach and Zündel. A similar mixture was previously recommended by the Prussian and Bavarian Governments. According to this  $16\frac{1}{2}$  lbs. of home-grown tobacco were boiled for half an hour in 55 gallons of water, and  $2\frac{1}{2}$  lbs. each of pure carbolic acid and potash added to the warm liquid (cost price, 11s.). This very effective dip is, however, troublesome to prepare, and may lead to poisoning of the sheep dipped therein. Instead of a decoction of tobacco-leaves, the tobacco lye might be used. Kayser recommends the substitution of nicotina, a species of tobacco-lye.

4. The **arsenic-dips** introduced by **Tessier** and **Matthieu** have met with an extraordinary diversity of appreciation ; some taking exception to their poisonous character, others Praising their great efficacy. But in many cases it seems that their poisonous action can be traced to careless application, especially just after shearing. The numerous small abrasions caused by clipping must all be thoroughly healed before dipping the animals, so that this should never be done until 8 to 14 days after they are shorn. Tessier's mixture consists of  $3\frac{1}{2}$  lbs. of arsenic and 22 lbs. of sulphate of iron, with 22 gallons of water. As modified by Trasbôt, Tessier's dip is made up of  $2\frac{1}{2}$  lbs. of arsenic, 11 lbs. of sulphate of zinc,  $17\frac{1}{2}$  oz. of aloes (all the weights quoted in this section are avoirdupois) and 22 gallons of water ; this formula is largely used in France. Matthieu uses alum instead of sulphate of iron ( $2\frac{1}{2}$  lbs. of arsenic,

22 lbs. of alum and 22 gallons of water). This arsenic and alum dip has been widely introduced in Würtemberg and Bavaria. Kehm employs 17½ oz. of arsenic, 13½ lbs. of alum and 22 gallons of water. It is important to observe that the arsenic is previously completely dissolved in boiling water to prevent any corrosive action. Putacher has also (verbally) recommended these arsenic-dips, but always precedes them with a cleansing bath of soft soap, and in severer cases has almost always found three dippings to be necessary, with an interval of nine days between each.

5. The **creolin-dip** recommended by Fröhner and specially prescribed by an order of the Prussian Government, dated 28th February, 1889, is carried out as follows :—If possible, the sheep are first shorn. This is not absolutely necessary with the creolin-dip; indeed, scabby sheep may be treated with good results in their wool and without any injury to the staple. After shearing, let all visibly scabby spots, especially the loins, back and neck, be rubbed for several successive days with a creolin-liniment, until the scabs are softened. This creolin-liniment consists of one part of creolin, one of spirit and eight parts of green soap. This embrocation forms an integral portion of the treatment (preparatory inunction). The sheep thus prepared are dipped twice in a watery solution (2½ per cent.) of creolin, an interval of seven days being left between each bath. The solution contains 1½ gallons of creolin to 55 gallons of water for 100 sheep, and is prepared by simply pouring the creolin into warm water 86° Fahr., and stirring. Each bath lasts three minutes, and after each such dip the animal must be vigorously scrubbed all over with brushes and then plunged again for a few seconds into the bath. The method of holding the sheep and all other manipulations are the same as in other forms of scab-dipping. Let there be no undue haste in the process, and give careful oversight to the workmen employed. The cost of each such bath is 8s., as against 11s. for the carbolic acid and tobacco-bath. A third dipping will only be necessary if the instructions given have been imperfectly obeyed. This creolin-dip is distinguished from other scab-dips by its certain and reliable action, and more especially by its non-poisonous nature and its cheapness (5½ gallons for 1s. 2½d.). Moreover, the wool is neither dyed by the creolin, nor in any way damaged, as is always the case with tobacco and arsenic. Of 440,000 sheep dipped in creolin in Prussia

during seven years, 76 per cent. were cured, a percentage which has risen in later years to 88.

Whether *Lysol*, which has been highly recommended by some, is equally efficacious with creolin still remains to be proved.

The method of washing in all scab-dipping is as follows:—After shearing, allow 3 or 4 days to elapse, which interval must with arsenic dips be extended to 8 or 14. The temperature of the bath must be about 86° Fahr.; for each sheep reckon from  $\frac{1}{2}$  to  $\frac{3}{4}$  of a gallon of liquid. Avoid the dip being either too cold or too hot. All the sheep in a flock must be dipped, including the apparently healthy; indeed, these should be taken first. The body must be completely immersed with exception of the eyes, nose and mouth, and remain immersed for three minutes. Let the dipping-tub be placed near the stable, in a cool, shady place, or, better still, partly sunk in the ground, and close at hand must be the scrubbing-tub. Each sheep must be dipped by two men, one holding all four feet, and the other the head, carefully protecting the eyes by pressing a thumb over each, and then plunging the animal in back downwards. Immediately after the bath it must be lifted into the scrubbing-tub, vigorously brushed, rubbed and its fleece run through with the fingers by two men, especial care being devoted to the back, loins and rump. Then plunge once more into the bath for a moment or two, and turn it loose into a *clean* stable or sunny pen-fold. Avoid the risk of its being wet by rain, as this not only brings danger of chill, but washes out the fluid and lessens its healing effect. It is understood that the whole operation must only be undertaken in warm weather. After the bath let all the stabling and shelters previously used be disinfected. A week later repeat the scab-bath, and again, if necessary, in fourteen days. Two dippings are in all cases indispensable. During the interval any freshly discovered scab-areas must be locally treated; and even when the whole dipping process is over, the flock should be carefully observed for some time. Finally, avoid as far as possible the routes and pastures previously used.

THE FOLLOWING ARE THE DIPS APPROVED OF BY THE BOARD OF AGRICULTURE  
AND FISHERIES IN THIS COUNTRY.

Sheep Scab Order of 1905, No. 6,879. Prescriptions for sheep dips approved by the Board for sheep-scab.

Quantities for 100 gallons of bath.

1. Lime and sulphur :—Mix 25 lbs. of sulphur with 12½ lbs. quick-lime. Triturate with water until a good thick cream without lumps is obtained. Transfer this to a boiler capable of boiling 20 gallons by the addition of water; boil and stir during half an hour.

The liquid should now be of a dark red colour; if yellowish continue the boiling until the dark red colour is obtained, keeping the volume at 20 gallons. After the liquid has cooled, decant it from any small quantity of insoluble residue, and make up the volume to 100 gallons with water.

2. Carbolic acid and soft soap :—Dissolve 5 lbs. of good soft soap, with gentle warming in 3 quarts of liquid carbolic acid (containing not less than 97 per cent. of real tar acid). Mix the liquid with enough water to make 100 gallons.

3. Tobacco and Sulphur :—Steep 35 lbs. of finely-ground tobacco (offal tobacco) in 21 gallons of water for four days. Strain off the liquid, and remove the last portions of the extract by pressing the residual tobacco. Mix the whole extract, and to it add 10 lbs. of flowers of sulphur. Stir the mixture well to secure an even admixture, and make the total bulk to 100 gallons with water.

Note.—The period of immersion in these dips should not be less than half a minute.

**Sarcoptic-Mange in Sheep.**—According to Gerlach's and our own observations, this occurs exclusively upon the portions of the head which are not covered with wool (called therefore *head-mange of sheep*), and only very rarely attacks the legs. Its favourite spots are the lips, corners of the mouth, ridge of the nose, chin, round about the eyes, also the cheeks and outer side of the auricles. Upon these we find firmly adherent, grey scabs, about  $\frac{1}{2}$  to  $\frac{1}{4}$  inch thick; and there is usually great itching. In warm weather the eruption spreads almost all over the head, retreating on the advent of a colder season. The ailment causes no further harm, and may be cured by creolin-liniment, or washing with creolin or strong tobacco-decoction. Artificial transmission to the woolly skin of a merino-sheep only produced a fleeting inflammation of the skin. A sarcoptic mange much resembling the mange of goats was observed by Roloff in sheep with fat buttocks (scabs as hard as stone and like scale-armour on the head and other parts of the body). Creosote or carbolic acid in glycerine were found to be the most efficient among mange remedies.

**Symbiotic-Mange in Sheep.**—This mange, which is also called the *foot-mange of sheep*, attacks the lower parts of the feet, and may even ascend to the testicles or udder. It is marked by itching, desquamation of epidermis and formation of scab.

## MANGE IN DOGS.

**Occurrence.**—This is a very common disease among dogs, especially in large cities. In Berlin nearly 10 per cent. of all sick dogs suffer from mange, and in some towns (Cologne, for instance) the percentage has been known to reach fifty. Dog-refuges, and breeding and training establishments have undoubtedly much to do with its dissemination.

**Symptoms.**—The mange of dogs (*sarcoptes squamiferus*) usually appears on the head, viz., on the ridge of the nose, at the root of the ears and on the eyebrows; it has also much liking for the belly, lower chest, the region of the elbows, the root of the tail and the paws. But the eruption may begin on other parts of the body, e.g., on the skin of the genitals. The mange generally spreads rapidly, so that after a month the whole body may be affected. Its aspect, however, varies much according to the power of resistance possessed by the skin.

Its first phenomena resemble flea-bites, being red spots, which are visible chiefly on the unpigmented skin, as well as on the tenderer skin of the belly and inner sides of the thighs. The surface is also often diffusely reddened owing to much rubbing and scratching. Upon the site of these, papules, varying in size from a millet-seed to a lentil, are soon perceptible, which may change into vesicles. These latter are often numerously scattered all over the body and, on bursting, leave large weeping places behind (so-called weeping mange). In other cases the process is quite dry, and turns to abundant desquamation of epidermis (so-called dry mange). More rarely the vesicles change into pustules, and this particularly on the finer skin of the lower belly and inner thighs, which pustules often show a dark centre the size of a pin-prick, and which is the seat of a mange-mite. Vesicles and pustules soon dry up into greyish-yellow crusts, while the upper skin scales off very much and the hair falls out, so that large bald patches appear. The skin itself thickens later, becomes wrinkled and creased and cracks are opened in it by the animal's rubbing. Most frequently we find the skin of head and neck folded into numerous wrinkles, which give the animals a very characteristic appearance. These skin-changes are accompanied by very intense itching, which increases in a warm room, near a fire or after any bodily exertion, and keeps the dog in a state of

constant unrest. If we scratch or rub the affected part the animal shows much delight and scrapes involuntarily with one or other of its feet. If the eruption last long and spread much, the dog grows very thin, and feeble animals may even perish.

**Diagnosis.**—The most important diagnostic signs of sarcoptic-mange are: Excessive itching, the frequent commencement of the evil on the head, its rapid spread, the ease with which it can be communicated to other dogs and even to men, the wrinkled appearance and baldness of the skin, the accumulation of scurf and scabs and the marked preference of the eruption for the ears, lower chest, lower belly, elbows and inner thighs. The surest of all diagnostic guides, discovery of the mite, is, however, generally very difficult. Owing to the depth at which it hides, it is effectually protected and can only be found by removing the deeper layers of skin with the scabs. To this end one must either scrape off the scabs with a sharp knife until they bleed, or remove smaller superficial portions of skin with scissors. Or we may try to tempt the mites from their lairs by heat, placing the dog in the front of a fire or covering it with a rug. In other cases, especially where the crusts become very thick, the mites are more easily demonstrable. As a last diagnostic resort one may bind a mange-scab on the human arm. Upon dead animals the mites may easily be discovered.

**Differential Diagnosis.**—Sarcoptic-mange is most frequently confused with the eruption of demodex. Both are infectious, both are accompanied by formation of nodules and baldness of skin and also with much desquamation of epidermis. The chief differences are that in sarcoptic-mange the itching is far more intense and that it is easier to cure; also that the demodex eruption usually displays a typical pustular character, and finally, that the parasites are easily found. An infectious skin-eruption, accompanied by intense itch and the other symptoms described, may always be regarded as mange when the results of microscopical investigation are negative. Sarcoptic-mange may also be mistaken for certain itching, papular eczemas. But the mistake is soon removed by the rapid healing of the latter. Widespread eczemas, which permanently vanish on a single application of mange remedies, must be presumed not to be sarcoptic-mange. In the latter disease

we also notice its communication to man, which fact may sometimes be of importance in distinguishing it from a simple eczema. Hundreds of men are every year so infected.

**Therapeutics.**—The prognosis in dog-mange is much more hopeful than in the mange of horses. But it must not be forgotten that the cure of long-standing and widespread mange in dogs is not always so easy, and that feeble and debilitated dogs frequently are unable to bear the treatment, and perish under it. But certain preliminary precautions are more necessary in dogs than in other domestic animals, as they must be prevented from licking off the drugs applied by tying up their mouths, muzzling them or covering them with leather wraps. The anti-parasitic remedies should also not be applied immediately after shearing, which, in long-haired animals, is indispensable. They should also not be laid on to weeping places of large area.

The following drugs are to be recommended in the mange of dogs :—  
 1. *Cresol*: We generally use a cresol-liniment which, according to the intensity and area of the eruption, we make of equal parts of cresol and green soap, with  $\frac{1}{2}$  to 10 parts of spirit. This liniment we rub in daily (in extensive mange one-third of the body daily); and a cure results on an average in two to three weeks. Also cresylic alcohol and spirit (1:10-20) and cresol-soap, as well as 2½ per cent. creolin-baths may be used.

2. *Tar*: Usually in the form of a tar-liniment (tar and soap equal parts, spirit, q. s.), and so applied that only one-third of the body is anointed at once. After remaining three days on the body, the liniment must all be washed off, and the dressing repeated. Cases of moderate severity are thus cured after three to four dressings.

3. *Creosote*, which is best mixed with olive or rape oil in the proportion of 1:10-15, and applied as above. With this remedy care must be taken to guard against poisoning.

4. *Carbolic acid*, as 2 to 5 per cent. carbolic oil or carbolic glycerine. With all three remedies it is well to leave a longer interval between each dressing, to prevent the risk of carbolic poisoning. By way of precaution a sulphate might be administered (sulphate of soda, to generate the harmless phenyl-sulphonic acid in the blood). Among other remedies we can recommend *Peruvian balsam*, which is particularly good for very delicate dogs and for treating the surroundings of the eyes. On

the other hand, we have only had very poor results from using naphthalin, which, moreover, smells badly. According to Siedam-grotzky, Tolu-balsam is also unreliable. Finally, we may mention as more or less useful remedies, benzol, naphthol, oxynaphthalin and ichthyol (Rabe, Friedberger). Benzine and petroleum are almost ineffectual.

Diet must be regulated during the whole time of treatment, and chills should be carefully avoided.

**Symbiotes on the Bodies of Dogs.**—The symbiotes mite of dogs, first discovered by Hering in 1836 and by him named "sarcoptes cynotis," is found sometimes in the purulent secretions of the otitis externa and also in the external ear-passages. That it can be brought into etiological connection with otitis is more than doubtful. Zürn and others believe in such a connection, basing their opinion on the processes in the ear-mange of cats. Nocard says that he has observed epileptiform spasms and deafness.

#### D.—THE MANGE OF CATTLE.

**I: Dermatodectic-mange.**—The favoured spots for this most frequent form of mange in cattle are the sides and hollow of the neck and the root of the tail. Thence it spreads over the back, the lateral thoracic walls, the neighbourhood of the horns, shoulders and even over the whole body. Its chief indications are: Nodules, exudation of fluid, increased desquamation of epidermis, loss of hair, great itching and a formation of very dry, greyish-brown scabs. The skin at last becomes thick and leathery, rough and wrinkled. Sometimes the disease is noticed to become more acute in winter, with a corresponding improvement in summer. As the eruption spreads over the body the animals grow thinner and sometimes die. Röll observed this form of mange in buffaloes. It appears that dermatodectic-mange occurs also along with symbiotic-mange. Treatment is the same as for other animals; thus, for example, creosote and decoctions of tobacco have been found very effectual.

**2. Symbiotic-mange.**—This form, which is also known as "*rump-mange*," occurs mostly in the hollows about the anus, as well as around the root of the tail, but may spread all over the body (back, neck, inner thighs) if due attention be not given to the skin. Such cases, nevertheless, are rare. It may also spread downwards to the lower parts of the feet (*foot-mange*). Its symptoms consist in deposition of scales

and scabs, loss of hair and itching ; but the eruption may last for years without further incommoding the animal or any serious spread ; it is only very slightly infectious. *Treatment* is only rarely needful ; as with horses, the milder mange-remedies suffice (carbolic-soap, creolin-ointment, tar-ointment).

**Sarcoptic-mange.**—This is extremely rare in cattle and is manifestly communicated by mangy horses. It represents therefore no independent form of mange and demands no further description, the symptoms being essentially the same as in horses. Neumann even doubts its existence.

**NOTE.**—There is no longer any doubt about sarcoptic-mange affecting the ox, numerous cases having been recorded during recent years.

#### E.—MANGE IN GOATS.

The **Sarcoptic-mange** of goats, which is the usual form of the disease in these animals, attacks at first only the lightly-haired places, more particularly the head (lips, ears, nose), but quickly spreads over the whole body. It is characterised by deposits of grey, bran-like scales, and of bluish-grey scabs, like those of fish, which are hard and fissured. The skin becomes thickened, cracked and wrinkled, grey and parchment-like, and shows bald patches, which gradually enlarge, so that the animals are often quite naked. There is also intense itching. Goat-mange does not seem to be at all rare, for in ten years there were nearly 12,000 cases in Austria alone. It is also frequently fatal to goats, and Wallraff noted 500 deaths out of 1,000 animals affected. The mite is very readily transmitted to man, and in one village Wallraff found nearly every family infected. Roloff also transmitted the mange to short-haired sheep (Somali) and to those with smooth wool and deficient in sweat (fat-rumped sheep). As regards *treatment*, baths should be avoided, as goats cannot bear them well. Use therefore creolin-ointment, tar-ointment, sulphur-salve, carbolic glycerine, etc.

By French observers (Mégnin and others), both a dermatodectic-mange (external ear-passages) and a symbiotic-mange (on the trunk) have been identified in goats.

#### F.—MANGE IN PIGS.

The **Sarcoptic-mange** of pigs (*Sarcoptes squamiferus*) is seen first on the head, especially in the hollows of the eyes, on the eyelids and cheeks, around the ears, and spreads thence

to the neck, back, inner sides of the thighs and finally over the whole body. At the outset we find these places covered with bran-like scales, blisters and pustules, and later with strong, whitish-grey or silvery scabs up to  $\frac{1}{2}$  inch in thickness, so that the animals look as if sprinkled with guano. Their skin is thick, wrinkled, very hard and firm. Their bristles also are loosened or fallen out, and sometimes stuck together in tufts. Beneath the scabs are mites, which, however, are difficult to find upon the living animal. *Treatment* consists in softening the scabs with soft-soap or glycerine, and in rubbing them with a tar or sulphur ointment.

In young pigs mange is sometimes called "sucking-pig rust," and generally represents a non-parasitic eczema (compare p. 435). That this "rust," as Sohnle believes, is always caused by sarcoptes-mites is improbable.

#### G.—MANGE IN CATS.

**Symptoms.**—The position most favoured by mange (*sarcoptes minor*) in cats is the head, which usually is alone attacked and especially the ears. At first red dots appear, also papules and blisters on the skin, which is then covered with small impetiginous scales. The hair becomes rumpled and partly falls out, and the upper skin desquamates freely. This scurfy matter, combined with the secretions from the papules and blisters, grows gradually to solid, greyish-yellow mange-scabs which measure up to  $\frac{1}{2}$  inch in thickness and contain a mass of mites. The underlying skin becomes hard and stiff, thickens and lies in folds. The eyelids often swell, and catarrhal and suppurative inflammation of connective tissue sets in. Itching is considerable, and the animals keep scratching themselves with their paws, shaking their heads and rubbing them against hard objects. Their general condition also is much deranged, even when the mange is strictly confined to the head. They are dull, grow thinner and not unfrequently perish. This eruption may also spread to the neck and entire body; when the paws are first affected, then the loins and flanks. In such cases death is much more likely to result. Transmission of cat-mange to mankind may also occur.

**Therapeutics.**—There is no great need for vigorous treatment; but baths, washings and embrocation of the whole body with oily remedies should be avoided. Carbolic acid, as being specially poisonous to cats, must never be used. As

the best and most harmless remedy we recommend Helmerich's sulphur-ointment. This is made up as follows : sulphur 2 parts ; carbonate of soda 1 part ; lard 16 parts. We have always effected a cure with this salve, provided the animal were not too much run down in condition. Not quite so harmless as this sulphur ointment is the otherwise excellent Peruvian balsam, after a limited application of which on the head we have at least a few times noted grave signs of brain irritation, and even decided maniacal symptoms with subsequent stupor and even death. The latter we have seen also after inunction with rancid cod-liver oil, a remedy recommended by some. Styrax appears to be somewhat less dangerous.

A *Sarcoptic-mange* on beasts of prey of feline species (lions and leopards) has been observed by Delafond and Bourguignon, and in more recent times by Johne. But this was not provoked by the *sarcoptes minor*, but by the larger burrowing-mite, *sarcoptes scabiei communis*. Its signs were at first circumscribed mange-patches on the breast, belly, back, etc., which speedily spread over the whole body, which became scabby, encrusted and almost hairless. The animals quickly grew thin, lost appetite and finally died. Sprinkling with Zündel's mange-dip resulted in fatal carbolic acid poisoning. The treatment adopted by Johne was an application of Peruvian balsam (1 : 3 spirit), the animals being introduced into a short and narrow cage, through the bars of which they were sprayed with Peruvian balsam and then vigorously scrubbed with brushes attached to long iron staves. After five such treatments, with intervals of eight to ten days, they were perfectly cured.

The so-called *ear-mange* of cats is caused by *symbotes auricularis*. The animal keeps shaking its head, holds it on one side and betrays symptoms of otorrhœa. We have found Peruvian balsam with an equal part of glycerine an excellent remedy, the ear-passages being previously cleansed.

#### H.—MANGE IN RABBITS.

I. **Sarcoptic-mange.**—The mange eruption produced by *sarcoptes minor* closely resembles cat-mange. It attacks chiefly the head (particularly nose, lips, brow), and is accompanied by formation of crusts and scurf, itching, thickening of skin, loss of hair, etc. Along with the head the ends of the extremities are generally also affected ; but the mange may spread all over, when, as a rule, the rabbit quickly grows thin and dies. A mange caused by *sarcoptes scabiei* also occurs on rabbits and sometimes covers the whole body and is communi-

cable to ferrets and guinea-pigs. As with cats, the *treatment* consists in rubbing with sulphur-ointment or Peruvian balsam.

2. So-called **ear-mange**, induced by symbiotes and *dermatodectes cuniculi*, is a somewhat common condition, consisting in inflammation of the covering of the external ear-passages, which develops into very thick layers of stratified, yellow-brownish, dry and fissured scabs, which sometimes completely fill up the ear-passages so that the auricle appears quite rigid. Zürn also observed violent inflammation of the middle ear and a dissemination of the *dermatodectes* mite over the whole surface of the skin. *Treatment* consists in softening and removing the scabs, and application by means of a brush of creolin-glycerine or carbolic-glycerine, or else creolin-oil or carbol-oil (2-5 per cent.), to the covering of the exterior ear-passages. Both these forms of mange, with the same localisation, occur in ferrets.

## FOLLICULAR MANGE.

**Occurrence.**—This eruption is most common on dogs and cats, rarer on pigs, cattle and goats; but the *acarus*-mite has been seen on sheep, the Sambur stag and finally on bats, rats, field-mice and earless marmots. It is also very frequently found in the comedones or dilated sebaceous glands of man, but without causing any injury.

**Natural History.**—The sac-mite, hair-sac mite, or *acarus folliculorum*, or *demodex folliculorum*, forms a species of the family of *dermatophili*, in the order of *acarina*, class *arachnoidea*, and belongs therefore to the group of arthropodes (articulated animals). It was first discovered by Henle and Simon in 1843, in the comedones of man, and, in the same year, by Tulk on dogs. Since then the following varieties have become known:—*acarus folliculorum canis*, *cati*, *suis* (*demodex phylloides*), *bovis* and *hominis*.

In its fully developed state the body of the *demodex folliculorum* of dogs is worm-shaped and lancet-like, but in pigs and cattle more resembles the form of a laurel-leaf. It is about  $\frac{1}{5}$  to  $\frac{1}{6}$  inch long and divided into three segments, head, thorax and abdomen, the two latter, however, being joined into one piece. The head is lyre or horseshoe-shaped and consists of various chitin-parts, which may be subdivided as follows:—

1. The front head, which forms the foundation of the entire apparatus for mastication; 2. a pair of upper jaws or mandiblæ; 3. a pair of clasp-like lower jaws or maxillæ; 4. the movable, many-jointed feelers or pedipalpi; 5. a single stiletto-like formation, being the so-called oral-valve. There are also two eyes in the form of unpigmented nodules on the sides of the anterior head. The thorax is short and bears in its two free edges four pairs of stumpy movable limbs, each made up of three parts, hip, thigh and terminal member, the latter of which has five chitin-pieces which give it rather the appearance of a paw. The lower surface of the breast displays a central ridge with four transverse ridges on each side. The long abdomen is also transversely striated, serrated along its edges and pointed behind. The young acarus has only three pairs of feet, is smaller and narrower and has no rings about its abdomen, and they hatch from spindle-shaped or oval ova. They pass through three stages of skin-formation before reaching maturity.

**Pathological and Anatomical Notes.**—Microscopical examination discovers the mites chiefly in the sebaceous glands, and more particularly round about the evacuatory exits, also in the upper parts of the hair-follicles, there being an average of 30 to 60 in each sebaceous gland. But this number may rise to 100 or even 200, so that the glands or their orifices quite bulge like voluminous bags. Along with these we often find a mass of eggs and larvæ. The mites lie in the glands almost without exception with their heads downwards. Around these bulging glands inflammation sets in, and white blood-corpuscles ooze out, so that the peri-glandular tissue becomes purulently broken down and, along with the gland, is changed into a purulent pustule. But these acne-pustules only develop when the mites are present in large numbers. When less numerous we only perceive atrophy and falling out of the hair.

The skin of the pig shows the most serious changes. Here several pustules run together and form large abscesses, which may contain as many as 1,000 mites, and cause deeply penetrating ulcers in the skin as large as half-a-crown. If the eruption last long, the skin becomes sclerotic and often three to four times its normal thickness.

**Symptoms in Dogs.**—The eruption caused by *demodex folliculorum* on dogs is very widespread and occurs in all breeds.

Of 70,000 dogs treated in the Berlin dog-hospital in nine years, not less than 1,200 (nearly 2 per cent.) had been attacked by acarus. Many animals show individual differences as regards the intensity and course of the complaint. With some the eruption remains for months confined to the same spot, while with others it spreads quickly over the whole body. Singularly enough, the complaint can only with difficulty be transmitted from dog to dog by inoculation, as compared with its evident contagiousness. Of many such attempts at transmission which have been made, almost all have resulted in failure, except a few observed by Hauber, Guinard and others. Neither can the disease be thus communicated to other animals, but a few cases of spontaneous transmission to man have, on the contrary, been observed. Its further spread upon the same animal results from rubbing and scratching, whereby the pustules are emptied and their contents, so to speak, sown over the body. Connected with this fact is also its quick dissemination after the earliest inunctions with medicaments. The aspect of the malady varies very much according to the numbers of mites present in the sebaceous glands.

1. *Squamous form of the acarus-eruption.*—In many cases, besides the loss of hair, one only finds an increased desquamation of epidermis, the surface of the skin being perfectly dry and showing no signs, or only very slight ones, of inflammation (*eczema squamosum*). This form occurs especially around the eyelids. At the same time the hair has mostly fallen out, leaving a bare circle about the eyes, the skin is slightly reddened and covered with cast-off epidermal scales. This limited appearance with such slight reaction of the skin makes the diagnosis of follicular-mange somewhat difficult, because its chief characteristics, the acne-nodules and acne-pustules, are absent. For this reason every case of the hair falling out, accompanied by slight skin-inflammation on the eyelids, should be microscopically examined for acari. But this kind of eruption is not rarely spread in patches all over the body, but usually without any itch. Its aspect has, however, an extraordinary resemblance to *tinea tonsurans*. Occasionally a darker pigmentation of the bald places is a characteristic sign of the disease.

2. *Pustular form of the acarus-eruption.*—More frequently the eruption manifests itself as an intense inflammation of the skin, with characteristic formation of papules and pustules, corresponding to the position of the sebaceous glands. This

form may also occur in circumscribed patches, especially on the head (ridge nose, lips, eyelids) and the paws, but may also spread all over the body. But it favours the head, throat, neck and inner sides of the limbs. At first the affected skin is swollen, higher in temperature and hyperæmic. The hair is ruffled, and scattered nodules reveal themselves, from a millet to a hemp-seed in size, which gradually change into bluish-red or purely suppurative pustules. The skin thickens, often to double its normal density, and is partially covered with greenish-yellow, sticky and purulent exudation, mingled here and there with blood; and partially with dried-up, greyish-yellow or blood-coloured crusts. The hair has partly fallen out and can be very easily pulled, when purulent matter often adheres to its lower ends. If the skin be laid in a fold and slightly pressed, blood is discharged and, on pressing more heavily, often a number of plugs of pus will follow. The degree of itching varies much in different individuals, being sometimes severe and sometimes almost absent; but in general it is much slighter than in sarcoptic-mange. If during its further progress the eruption be not altered by the animal's scratching and rubbing, the skin will gradually dry up, but remains thicker and disposed to lie in folds, showing several hairless or very scantily haired patches, on which, in the latter case, the hair is ruffed and strikingly lustreless. The skin is also covered with thick layers of blue-grey epidermal scales and scabs, between which are fissures and cracks which easily bleed. It thus gains an appearance like that of the bark of a tree or an elephant's hide, and the head particularly reminds one of a wart-hog (*phacochoerus*). The surface of the skin is also rough and covered with lumpy, hard, nodular excrescences, from which further pus-plugs may be squeezed. The unpigmented parts of the skin are of copper-red colour, or else display upon a slightly reddened ground bluish-red, knotty protuberances (hence the earlier name of "red-mange"). Finally, we find in the upper skin-layers, corresponding to the situation of the suppurated sebaceous glands and hair-follicles, pitted hollows, with proliferations of connective tissue resembling papillæ. In spite of these serious changes, the animal's general condition is often very little disturbed, there being nothing beyond a little chill and slight loss of flesh; its appetite, indeed, is usually increased, so that it often seems to be really ravenous.

This aspect of follicular-mange is, however, gravely altered if the animals gnaw or rub much. The affected spots are then

changed into hairless sores, which secrete pus, or even into genuine skin-ulcers. At the same time quite enormous swellings of certain parts, such as the head, round the eyes and neck, arise as a result of inflammatory oedema of the subcutaneous connective tissue. In this condition the animals rapidly grow thin, emit an unpleasant, sweetish-putrid smell and present a wretched appearance. As a rule they die then either from exhaustion or of pronounced septicæmia.

**Symptoms in Pigs.**—The phenomena of the skin-complaint produced in pigs by *demodex phylloides* are, according to Csokor, protuberances, varying from a grain of sand to a hazelnut in size, out of which small tumours at first develop, which grow larger and become at last broad skin-ulcers. As an intermediate stage, pock-like lesions are mentioned by some observers, which may give rise to confusion of this eruption with swine-pox. Only the softer-skin parts are attacked, such as the vicinity of the snout, neck, lower breast, flanks, groin, belly, and especially the inner sides of the thighs, while the upper head, back and outer sides of the limbs escape. The matter which can be squeezed out of the tumours contains from 50 to 100 mites, with their various larval conditions and cast-off husks, while the larger abscesses shelter from 500 to 1,000. This mite is twice as broad as the *demodex folliculorum* of dogs. The eruption is much more contagious than that of dogs.

**Symptoms in Other Animals.**—Grimm and Oehl have noted cases in cattle in which the skin of the entire body, excepting only the head and extremities, was sprinkled with nodules from the size of a pea to a hazelnut, which were most abundant on the neck and shoulders. These nodes were hairless, smooth of surface, and from them a thick, tough pus could be pressed, in which very large number of demodecides could be found. Other parts only showed redness, slight swelling, loss of hair and desquamation of epidermis. Oehl states that cattle recovered spontaneously, but a cure was hastened by rubbing, in a 10 per cent. creolin ointment.

Upon goats Bach noticed about 40 such nodes, from a pea to a cherry in size, with dough-like, greyish-yellow contents, and especially on the shoulders and about the flanks. These nodules contained each up to 1,000 acari. There was moderately severe itching and great loss of condition. A treatment extending over 30 days proved to be of no avail.

In cats the *demodex* eruption is seated chiefly upon the head (nose, eyes, ears); the clinical aspect is similar to that in dogs.

**Diagnosis.**—In many cases the evidence of the characteristic bluish-red pustules, combined with localisation of the malady upon the head or extremities, suffices for diagnosis even on a superficial examination. But certainty can only be secured in the squamous form of follicular-mange by microscopical investigation, which must be carried out by thinly spreading some matter squeezed from a pustule upon the object-glass of a medium-power microscope. If there are no pustules, scrape the skin superficially and place the scrapings, or the roots of a few plucked-out hairs, under your glass. The ailment may be *mistaken for*: 1. non-parasitic, impetiginous eczema and traumatic suppurative dermatitis; 2. for the pustular exanthema of distemper in dogs; 3. for sarcoptic-mange; 4. for inflammation of the eyelids (Blepharitis); 5. for tinea tonsurans; and 6. for non-parasitic acne and furunculosis.

**Prognosis.**—In a few cases it is said that spontaneous cure may occur, but these are certainly rare. In general the forecast must be unfavourable. When widely diffused, the malady has so far been only seldom cured, even after months of persistent treatment. On the other hand, apparent cures, followed later by relapse, have not unfrequently been published as genuine. In any case it is necessary carefully to watch dogs that seem to recover for weeks and months, before one can be sure of the result, and it is very easy to be deceived in this matter. Even when the eruption is locally restricted, one must be very cautious in forming a forecast. A cure is then certainly easier, but the complaint may notwithstanding spread very rapidly as a direct result of the treatment. One ought always to warn owners from the very first of the great obstinacy of the disease. When the acarus eruption is widely spread, it is better to kill the dog at once. But the individuality of the various animals much affects prognosis.

**Therapeutics.**—The number of remedies in use for follicular-mange is extraordinarily great, and well illustrates the helplessness of therapeutists. So far the following have been recommended: The various carbol-preparations, creolin, benzol and petroleum, Peruvian balsam and styrax, mercurial mixtures, cantharides-ointment, sulphuret of potash, naphthalin, naphthol, strong coal-tar oil, nitric acid, tincture of iodine, Lugol's solution, sozoiodol, ethereal oil of juniper berries, sulphur-ointments, etc. According to our own experience,

those which have best stood the test of experiment are: Peruvian balsam, cantharides-ointment in combination with sulphuret of potassium, sublimate-ointment and also inunction with concentrated creolin.

1. *Peruvian balsam* is especially recommended by Siedam-grotzky. He cured a dog which was affected all over after a two-months' daily anointing with it and carefully squeezing out the pustules, curing not only the local sores, but the entire eruption. We also have cured slighter cases, but only after prolonged treatment, in one instance extending over 58 days. In another case of a badly diseased animal we obtained a great improvement, which looked like complete cure, after 16 weeks of treatment, but on cessation of this the eruption soon reappeared. Certainly very long and persistent treatment with Peruvian balsam is required to effect a complete cure. The remedy, moreover, is very costly, and renders the symptoms more severe at first.

2. The plan adopted by Brusasco in Turin consists in washing the affected skin with a solution of *sulphuret of potassium* ( $1\frac{1}{2}$  drams in  $1\frac{1}{2}$  pints of water) and subsequent inunction with dilute cantharides-ointment. We also achieved complete cure in very advanced cases in a relatively short time. But care must be taken not to touch the conjunctiva of the eyes with the cantharides, or suppurative panophthalmia may set in, which may even prove fatal. We would therefore advise treatment of that region with Peruvian balsam. The process is best carried out by first clipping the animal (if the eruption be widespread) and bathing it completely in sulphuret of potassium (say 7 oz. to  $15\frac{1}{2}$  gals. of water for a large dog), and then on the next day rubbing one-third of its body thoroughly with the dilute cantharides-ointment, and another third part on the next and following days. One or two days later wash it all well off with the solution of sulphuret of potash; and then, after a few days' pause, recommence anew, continuing thus until a complete cure is effected. But that even this plan may fail is proved by the case of a Pomeranian dog with widespread eruption which we did not succeed in curing after 68 days' treatment, in which it was 12 times bathed, 9 times anointed all over with salve, besides receiving 9 separate local applications of the cantharides-ointment. But we must add that a further 16 weeks of treatment with Peruvian balsam had no better result.

3. Inunctions with 1 per cent. *sublimate-ointment*, along with

washings of the same drug in a 1 per cent. solution, are also, according to our experience, a method that meets with success if long persisted in. But the dogs must be carefully hindered from licking off the salve, or they may easily be poisoned. This, therefore, is one of the most dangerous of remedies.

4. Rubbing with *creolin* in an equal quantity of spirit, combined with creolin-baths (2 per cent.), has also wrought a cure for us in a few not too advanced cases.

These four remedies may either be applied singly or one after the other, but must always be used with care and perseverance. The rubbings must be daily and should be accompanied by careful pressing out of all newly-formed pustules, the hair should be clipped from time to time, and the dog well fed. None of the other remedies mentioned above seem to be reliable in any but slight attacks. Better, perhaps, may be the *baths of sulphuret of potash* recommended by Mégnin, which he used daily for two months for fifteen minutes at a time. The sulphuretted hydrogen hereby developed is a deadly poison for the mites, which explains largely its favourable result, as it does also in Brusasco's process.

Prophylaxis consists in separating sick from healthy dogs and in thorough disinfection of their beds. This is a disease which ought to be legally proscribed, and all dogs suffering from it promptly killed. It can never otherwise be extirpated.

#### SKIN DISEASES PRODUCED BY FILARIA.

I. Among *horses and asses* we find a peculiar, nodular skin affection which is caused by round worms (*filaria haemorrhagica s. multipapillosa*), and which is known as *dermatitis pruriginosa* (Rivolta) and *dermatitis granulosa* (Laulanié). According to observations made by the latter, the skin is in this disease permeated by numerous nodules of spherical or elliptical shape, which penetrate to its subcutaneous cellular tissue. The centre of each is soft and caseous and contains a round, spirally-coiled worm with characteristic transverse striation, which in the older nodules may be already decomposed. The skin itself is thickened. The centre of the nodule has plainly lost its capsule and can be peeled off; the process begins with small-cellular infiltration of the cutis and subcutis. Laulaníe believes that the parasite can remain for years in the nodule, and has found that the nodular eruption retrogrades in winter and develops afresh in summer owing to the freer flow of blood

to the skin. According to Railliet and Moussu, the filaria inhabit the subcutaneous, intra-muscular and intra-fascicular connective tissue and penetrate from the subcutis to the skin. When the nodules burst they retire more deeply in. Contamine and Drouilly observed bleeding of the skin and haemorrhagic skin-nodules in Hungarian horses, which were produced by the incursion of filaria haemorrhagica,  $2\frac{1}{2}$  to  $2\frac{3}{4}$  inches long. Rivolta considers that the removal of the affected tissue is necessary for a cure. It is probable that many of the so-called "tubercular eruptions" described by earlier authorities, and especially by Haubner, belong to the chapter of granular dermatitis produced by nematodes.

2. In dogs Siedamgrotzky and Schneider observed a pustular eruption on the outer surfaces of both fore and hind legs which was caused by round worms. The small, intensely itching pustules and nodules, surrounded by a reddened area, were situated upon hairless parts and each contained one to three round-worm embryos, with a bodkin-shaped tail and from  $\frac{1}{16}$  to  $\frac{1}{8}$  inch in length, which had evidently wandered from the hair-sacs. They were to be found among the straw bedding along with full-grown ones  $\frac{1}{8}$  inch long. Recovery followed without medical treatment within three weeks. Rivolta also found filaria in ulcerative skin eruptions of dogs.

**Filaria medinensis.**—The medina-filaria found in Egypt, India and other southern lands produce round worms, which grow to be  $\frac{1}{16}$  inch in diameter and usually live as parasites in the subcutis of man and the domestic animals, where they cause local swellings and abscesses in the extremities. They are most common on cattle, dogs and horses. Where, as on man, generally only one filaria is found, one may meet with two, three, four and even five on the domestic animals.

Filaria irritans produces "summer sore" of horses.

- SKIN DISEASES CAUSED BY FLIES, GNATS, GAD-FLIES AND THEIR LARVÆ.

**General Notes.**—Our domestic animals are plagued by the above-named insects in many ways. Some cause no real skin affection, but merely irritate the animals, prevent them from feeding and cause waste of fodder. Among such are the common house-fly (*musca domestica*) and, as a sweat-sucking fly, the raven-fly (*musca corvina*). Another section settle temporarily on domestic animals and suck blood, especially on tenderer parts of their skin. Such are the gad-fly of cattle (*tabanus bovinus*), the rain gad-fly (*tabanus s. hematopota pluvialis*),

the blind gad-fly (*tabanus s. chrysops cæcutiens*), mosquitos, tsetse flies, etc. Others settle upon the animals in great numbers, and by their many stings cause serious inflammation. Such are the common stinging gnat (*culex pipiens*), the common stinging fly (*stomoxyx calcitrans*), the storm-fly (*anthomyia meteorica*), the common and ornamented crawling gnat (*simulium reptans and ornata*) and, finally, the most dangerous of all gnats, the Columbacz gnat (*simulium Columbaczensis*). Many insects require also to be considered because their larvæ (maggots, mites) cause trouble on, in or even under the skin. Thus the blue blow-fly (*musca s. Calliphora vomitoria*) and the carrion-fly (*musca cadaverina*) lay their eggs in wounds and ulcers, as does also the grey meat-fly (*sarcophaga carnaria*). To this category belongs finally the most important of all, clinically considered, viz., the gad-fly (*aestrus bovis*), whose larvæ are found in the subcutaneous connective tissue. Among the above those of chief pathological interest are :—

I. The **Columbacz gnat** (*simulium Columbaczensis*, or *maculata*), an ash-grey insect about the size of a flea, whose ravages were first noticed in the neighbourhood of the Castle of Columbacz, near Temesvar, in Hungary, where in 1783 nearly 600 animals (horses, cattle, pigs, sheep) died from its stings. It is also found in Austria, Bohemia, Prussia and in scattered spots all over Germany. The insects breed in swarms after floods, forming thick blackish-grey clouds, which settle down on animals grazing in the open, so that white cows appear quite black. The animals become frantically restless, rush off home, fling themselves on the ground, groan and bellow. On examination the gnats are found, not only on the hair, but buried in all the natural openings of the body, and, as a result of their stings, painful swellings arise all over the body, and especially on the mucous membranes of the nose, mouth, eyes and vagina. The animals may perish in a few hours from suffocation produced by the swelling-up of their breathing passages. Those less severely attacked are ill for several days, eat little or nothing, and show next day a number of small, hard, painful nodules on the skin, which do not pass away for 3 to 4 weeks, and also various serious swellings of the mucous membranes. These inflammatory signs often remain for fourteen days or longer. On *post-mortem* examination of the fallen animals, we find intense, gelatinous and bloody infiltration of the subcutis, swelling and inflammation of the visible mucous membranes, as well as signs of hyperæmia

and oedema of the lungs. Occasionally the insects have penetrated to the throat and larynx, or even into the wind-pipe, setting up inflammation and swelling there and even stopping up the lumen of the trachea. *Treatment* of the sick animals is restricted to cold washings and douches and scraping the swollen membranes until they bleed. Eventually tracheotomy has to be performed. As a *prophylaxis* washings with tobacco-lye are recommended; but all the fly-remedies may be tried, such as creolin, carbolic acid, tar, naphthalin, petroleum, stinking animal oil, asafoetida, decoctions of walnut-leaves in vinegar, etc.

A similar case of wholesale attack on cattle, horses, and sheep, caused by *simulium ornata*, is reported by Müller, when, out of 170 cattle attacked, 26 died.

2. The **skin gad-fly** (*oestrus sive hypoderma bovis* and *hypoderma lineata*) produces what are known as gad-fly boils (warbles), which are most troublesome in cattle, but are rarer in horses, asses and sheep, affecting chiefly the shoulders, loins, flanks and breast. These are caused by the female gad-fly laying her eggs upon the ox's skin, where they hatch, and the larvæ bore through the entire thickness of the skin and settle in the subcutaneous cellular tissue, where their further development takes place. This lasts about nine months (July to March). They produce inflammation and suppuration round about them and finally a tumour as large as a pigeon's egg (warbles). When the larvæ are ripe, they quit their lurking places after first widening the entrances with their hinder parts. The number of such boils on the same animal is usually small, but there may at times be 50 or even 100 on the same skin, producing a very injurious effect upon the animal, which rapidly loses flesh. On the approach of the flies the cattle begin "gadding," grow restless, spring wildly hither and thither, bellow loudly, elevate their tails and seek if possible a refuge in water or in their byres. The *treatment* for warbles consists in squeezing out the riper larvæ, or opening them with a knife; and *prophylaxis* in giving attention to the skin, removing the eggs and rubbing the upper portions of the body with creolin, tar, dilute carbolic acid, solution of asafoetida or a decoction of walnut-leaves. Do this especially on sultry days which threaten thunder, and before sending the animals out to graze. It is also recommended to rub their backs with salt or rancid butter during the fly-season.

Hinrichsen believes that he has found the larvæ of the gad-fly in the form of fine parasites in the canal of the spinal cord in cattle. It

is his opinion that the cattle take up the gad-fly eggs with the herbage, and that the young larvae penetrate from the stomach and intestines to the spinal canal, dwell there for five to six months and then migrate through the inter-vertebral foramina as far as just below the skin. According to Hinrichsen, these larvae are to be found in the spinal canal of from 40 to 50 per cent. of all cattle that have been out at pasture especially during the months December to March. Ruser and Horne also confirm the frequent presence of larvae in the spinal canal, but the latter, in opposition to Hinrichsen, supposes that they enter through the skin.

A skin disease very similar to gad-fly boils has been observed by Railliet in dogs, but caused by the larvae of *ochromyia anthropophaga* (possibly larvae of the human gad-fly, *dermatobia noxialis*?) penetrating beneath the skin. Its symptoms were hard nodules, the size of a lentil and painful when touched, with a cylindrical opening in the middle. After six or seven days the nodules grow softer and enlarge to the size of a small nut, when the larvae emerge. These nodules may occur anywhere, but seem to prefer the tail, ears and paws. Here, too, removal of the larvae is the only cure. In horses also the larvae of flies are found in the skin (*hypoderma equi*).

3. The so-called **fly-larvae sickness** of lambs is known especially in Holland, and is caused by the maggots of *musca Cæsar* (*lucilia Cæsar*, *L. sericata*, *L. macellaria*). The flies lay their eggs between the hind legs of lambs suffering from diarrhoea. The larvae are said to seek out the more woolly parts of the tail root and loins, where they vegetate in clusters under the wool, perforating the skin like a sieve. Older animals are not often attacked. Treatment consists in removal of the maggots and application of carbolic solution, decoction of tobacco, etc. Certain blue-bottle flies seem to be of special interest in tropical countries. According to the reports of Frantzius (Virchow's "Archives," vol. 43), thousands of calves perish there owing to the maggots laid in their navels and the consequent local inflammation.

The remedies which may be applied to the skin as a protection against winged insects are: rubbings with highly odorous fluids, such as creolin (2 per cent.), asafoetida, benzine, petroleum, stinking animal-oil, laurel-oil and decoctions of tobacco and walnut-leaves; also with bitter solutions such as decoctions of absinthe, gentian, nux-vomica or tanacetum. The alcoholic extract of Persian insect powder is also said to be very good.

#### LICE (HÆMATOPINUS) AND TRICHOECTES.

I. **Lice** (*Hematopinus*) are blood-sucking parasites, which attach their eggs (nits) to the hairs of the animals they infest.

Most domestic animals have their special species, thus the *horse* has the *haematopinus* or *pediculus equi*, or *macrocephalus*, the *ox* has its *haematopinus eurysternus* and *tenuirostris*, the *pig* its *haematopinus suis* or *urius*, the *dog* is infested by *haematopinus piliferus*, the *goat* by *haematopinus stenopsis* and the *rabbit* by *haematopinus ventricosus*. The places most favoured by these parasites are the neck and back, also the root of the tail, in cattle the vicinity of the horns, and in pigs the inner sides of the hind thighs. Lice are most common on cattle, and are found even on the best nourished animals; the same may be said also of dogs. But in general they thrive best on poor and ill-fed animals, and their presence is always an indication of a neglected skin. The phenomena produced by lice are itching, loss of hair, marked desquamation of epidermis and also numerous nits or eggs attached to the hairs. Moreover, the animals often diffuse an unpleasant smell, and are (especially dogs) covered all over with a greasy or impetiginous matter.

2. **Trichodectes** are not blood-suckers, but *epidermis-eaters*. In contrast to the pointed head of the *haematopinus*, theirs is broad and shield-shaped; they live on epidermal-scales and hair, and their favourite haunts are the head, neck and legs of domestic animals. We distinguish the following varieties of the parasite: on the *dog* *trichodectes latus*, on the *horse* *T. pilosus*, on the *ox* *T. scalaris*, on the *cat* *T. subrostratus*, on the *goat* *T. climax*, and on the *sheep* *T. sphærocephalus*. The latter causes very intense itching in sheep, which may arouse suspicions of scab.

The following *drugs* are highly spoken of as remedies:—  
 1. *Grey mercurial ointment*, which may be applied in small doses on all domestic animals, especially horses, pigs and dogs. Even on full-grown cattle, which are known to be very sensitive to mercury-ointment, we have had capital results from rubbing about four scruples of the grey salve between the horns, without noticing any poisonous effect. For dogs it is best to smear the ointment under the collar in quantities about as large as a pea or a bean. We have also obtained extraordinarily good results in dogs from carefully washing them with one per cent. sublimate solution, doing only a small part at once and being particular to rub it quite dry afterwards.  
 2. *Persian insect powder*, which should be sprinkled between the previously moistened hair, is not strong enough for the larger animals.  
 3. *Tobacco-decoctions*, 4 to 5 per cent., are

very good for sheep and dogs, but less to be recommended for horses and cattle; nicotina, a kind of tobacco-lye, is also well spoken of. 4. *Creolin* is especially commended by Grodts for lice on horses. He applies it in a five per cent. solution with brushes, repeating the dose in six days and again in another six. Each such washing must be accompanied by a careful cleansing of the stable. Among the many other remedies we may enumerate lysol, benzine, staphisagriae in vinegar (1 : 20); a mixture of powdered cevadilla-seed, staphisagriae and white hellebore in equal parts with two parts of aniseed; this, however, is only suitable for the larger animals; benzol, diluted with green soap, spirit or oil; carbolic acid in a five per cent. solution; and also for fancy bred dogs etherial oil of aniseed, mixed with ordinary oil or dissolved in spirits (1 : 10). Arsenic lotions (arsenic and potash, each  $\frac{1}{2}$  oz., water and vinegar each 3 pints), as recommended by Viborg and Schleg, must be used with caution, but are especially efficient on large animals with many lice. The decoction of nux-vomica suggested by Eck must be rejected as far too dangerous, and the poisonous foetid animal oil can be well replaced by creolin.

For trichodectes in sheep Thierry uses a mixture of powdered cevadilla-seed and Persian insect-powder. To destroy their eggs washings of vinegar may be applied (unless one prefer to await their development); this destroys them by dissolving their calcareous shells. Finally, and in obstinate cases, we recommend cutting off the hair and disinfecting the stables.

#### VARIOUS OTHER SKIN-PARASITES.

I. **Fleas** occur on dogs (*pulex canis*), cats (*pulex felis*), and on rabbits (*pulex gonicephalus*). They are blood-suckers, which not only irritate their hosts with great itching, but also give rise to chronic skin-eruptions. The skin is, moreover, dirtied by the dung of the fleas, which is often found thickly scattered among the hair like coffee-grounds. The best remedy for fleas is the Persian insect-powder (*flores pyrethri*), which should be dusted over the skin, after previously slightly damping it in order that it may adhere better. It is also advisable to let the dog remain out of doors for a time after sprinkling, lest the cast off fleas find a lodgment in the stable or house. Also washing them with a two per cent. solution of creolin is a very effective cure. A very troublesome parasite for domestic animals in America and Africa is the sand-flea (*sarcopsylla penetrans*).

2. **Ticks** are large insects resembling mites or acari, which live in woods, bushes and grass, whence they attack animals and bore into their skin in order to suck blood. We distinguish the dog-tick (*ixodes ricinus*), which is found on cattle and sheep as well as on dogs; and the ox-tick (*ixodes reticulatus*), which settles as a parasite upon the skin of cattle and sheep. The American tick (*ixodes Americanus*) is said especially to attack horses. According to Janson, ticks are a positive danger to horses in Japan, where many succumb yearly to their attacks. Respecting the cattle-ticks (*boophilus bovis*, *ixodes annulatus*), which stand etiologically related to Texas fever, compare the chapter on Texas fever in Vol. I. Treatment consists in the application of common fatty oil, creolin-oil, carbolic oil, or of the etherial oils (turpentine, aniseed, caraway and fennel oils), or of tobacco-decoctions, petroleum, etc. These should be put on to the tick as found with its head inserted into the skin. It is not well to pluck it off violently, as the head then remains behind in the skin.

3. **Louse-flies** (*pupipara*) occur as parasites on horses (*hippobosca equina*) and upon sheep (*melophagus ovinus*, sheep ticks). The louse-fly of horses settles about the tail and anus and upon the lower belly, and excites chiefly by its rapid running about rather than by biting or sucking, rendering the animal very restless. The louse-fly of sheep occurs chiefly upon grazing sheep, of whom it sucks the blood. It excites intense itching, which makes the animal rub itself and damage its wool. The excrements of these flies often dye the wool green. Remedies for sheep are tobacco-decoctions, washings with solutions of creolin or of carbolic acid, as well as grey mercury-ointment, 2 to 3 scr. each rubbed along the back and under the neck, and all such treatment is most efficacious after shearing.

4. The **bird-mite** (*dermanyssus avium*) infects especially fowls, pigeons, swallows and cage-birds, as well as their roosts, nests and cages. The colour of these mites is blood-red or reddish-brown, and at night they betake themselves to any animals that may be near (cattle, horses, dogs and cats), upon whom they may permanently settle, causing much itching and an eruption of the skin. Thus Steinbach noticed a mange-like eruption on horses, with isolated scabs and fissures and accompanied by intense itching. The mites were found in the forelock and mane and at the root of the tail. Möbius also saw a

similar skin affection on eight cows, with round hairless patches, which was vainly treated for two years, and only disappeared on the removal of certain swallows' nests in which the bird-mites had dwelt. Prietsch found a great number of sore spots on two horses, viz., on their necks and backs, which were devoid of hair and partly covered with bran-like scales, and partly with thin scurf. The itching was considerable, especially towards evening and in the night. According to Trasböt, these mites prefer to settle on horses underneath their cloth cover. Moreover, these bird-mites, by settling in the outer ear-passages of cattle, may produce an otitis externa (Gassner) and, by penetrating to the middle ear, give rise to peculiar signs of cerebral irritation. Thus Stadler and Schuemacher noted in cattle slaughtered on account of mania that the inner parts of the ear were quite choked with these mites. (Compare also the skin diseases of birds.) In the ear-passage of a cow which used to shake its head for hours together, Ostertag found an allied form of mite, viz., *gamasus auris*. Treatment consists in removing the hen-roosts, swallows' nests, etc., from the neighbourhood of the cattle, and in the application of mite-killing remedies (creolin, carbolic acid, etc.). A thorough examination and cleansing of the byres is also advisable.

5. The autumn grass-mite (*leptus autumnalis*) lives in dry grass, among the ripe corn, and also on lilac and currant bushes, whence it travels to the backs of horses and dogs, causing a pustular skin-eruption, chiefly on the head, round the mouth, ears and eyelids, and also upon the inner sides of the hind legs and on the genitals. The local changes are at first circumscribed red spots, from a poppy-seed to a hemp-seed in size, which on closer examination are found to be papules or pustules, on which rest a number of the r̄-coloured mites. On pressure, a bloody fluid can easily be squeezed out of them. Later these spots enlarge to the size of a shilling, are bare or but sparsely haired, the skin becoming hyperæmic and sensitive, and the hair falling out. There is usually also a moderately severe itch. This eruption may last for several weeks, but can easily be removed by rubbing with creolin or carbolic ointment, or with creolin or carbolic glycerine. Eloire has also found the mite on fowls. According to Mégnin, the autumn grass-mite is the young form (the six-legged larva) of the velvet-mite, *trombiculum holosericum*. It is also supposed to cause the vesicular eczema of the rape-sickness, and also other skin eruptions on

dogs, cattle and horses, which are accompanied by severe itching. It is said to be met with on the leaves of buck-wheat.

## APPENDIX.

### SKIN DISEASES OF POULTRY PRODUCED BY ANIMAL PARASITES. THE MANGE OF BIRDS.

Among the multitude of animal ecto-parasites which infest our domestic poultry, the mange-mites play a chief part, and may be divided into two chief kinds :—

1. *Dermatoryctes mutans* (Ehlers), a mite resembling sarcoptes, which was formerly called sarcoptes mutans (Robin), sarcoptes avium (Gerlach), or cnemidocoptes viviparus (Fürstenberg).

2. A mite discovered by Caparini (*dermatophagus gallinorum*), which produces a dermatophagous or symbiotic mange. According to Troussart and Neumann, this mite is identical with epidermoptes bilobatus and bifurcatus (Rivolta).

1. **Dermatoryctes-mange** is caused by a mite very like the sarcoptes-mite of mammalia, but not identical therewith, and which is distinguished from a sarcopt by the structure of its cutting jaws. The females are larger than the males (0.4 mm. long) and are almost spherical in shape. The males are more oval (0.2 mm. long). These dirty-yellow mites possess four pairs of feet when mature and only three when in the larval state ; on the anus grow two long hairs. The dermatoryctes mite, as its name indicates, is a burrowing insect, which bores tunnels under the epidermis. Fowls are most subject to this mange, and imported breeds more so than native ones. But the mite can pass to other birds, such as pigeons or exotic birds, and Mégnin has found it on turkeys, pheasants and singing birds. Ostertag has observed its passage to a horse. According to Railliet this mange is only slightly contagious.

The *symptoms* of this gallinaceous disease, which is also known in Germany as "chalky legs," and also as the "fish-scale sickness" (ichthyosis) of fowls, are as follows :—The mange settles mainly on the legs, but may obviously be transferred by the bird's scratching and pecking to the head (comb, wattles, beak). At first we notice upon the front surface of the extremities (corresponding to the metatarsal and adjoining bones) small, whitish-grey spots, which enlarge and grow to

round or zig-zag crusts. Both the front and back surfaces are later covered with uneven, lumpy, yellowish-grey scabs, which are very porous and cracked, may become  $\frac{1}{2}$  inch thick, and are of foliated structure, some of the leaves having a ready shine like mother-of-pearl, and feeling greasy to the touch. By this scabby incrustation the legs are thickened to deformity, the horny foot-scales become detached and stand out, and the skin appears as though coated with chalk or lime (so-called chalky leg). The scabs consist mainly of epidermal-scales, dry products of inflammation and mites, and beneath them the skin is red, swollen, purulently infiltrated and easily bleeds. There is usually moderately severe itching, and the birds peck at the scabs and constantly rub their legs. In severe forms of the disease they cannot run and can scarcely stand, but squat upon their breasts. Later emaciation and cachexia show themselves, and in very severe cases death even may result.

The treatment consists in removing the scabs after a preliminary softening with glycerine or soft-soap, and in rubbing with ointment of creolin, carbol or creosote (1 : 10 to 20), tar, Peruvian balsam, styrax, Helmerich's sulphur-salve, etc. At the same time sheds and roosting-places must all be disinfected.

2. The **Symbiotic-mange** of poultry attacks especially the neck and breast, but may quickly spread all over, including the comb and wattles. The skin becomes covered with thin, transparent, straw-coloured epidermal-scales, which form in layers one on the other, and may grow to thick, dry, doughy scabs of a dirty yellow-grey colour. The skin beneath is hyperæmic, and there is no itching. This symbiotic-mange quickly leads to loss both of flesh and strength, and not rarely to death. Treatment is the same as for the preceding form of mange. Friedberger noticed a complication of dermatoryctes and symbiotic-mange upon the same animal.

Under the name of **Sarcoptes cysticola** (*laminoscoptes gallinarum* s. *cysticola*, *symplectoptes*, or connective-tissue mite) a mite resembling a sarcopt has been described by several writers. It is discovered enclosed in a capsule, in the form of a whitish or yellowish, oval nodule, in the skin, the subcutaneous cellular tissue, the inter-muscular connective tissue, as well as upon the serous membranes of the organs in the abdominal cavity. It produces sometimes a mange-like eruption upon the skin (Unterberger).

VARIOUS OTHER SKIN DISEASES OF POULTRY CAUSED BY ANIMAL PARASITES.

Of the various animal parasites which inhabit the skins of poultry, and cause inflammation there or injury of plumage, we may mention the following :—

1. The **demodex of the tame pigeon** (*harpiphyynchus nidulans*) lives in the feather-sacs, which are thus expanded into yellow capsules or cysts as large as a pea or even a bean ; also in the connective tissue cysts of the skin, particularly on the lower breast and beneath the wings. Their knob-like protuberances consist almost entirely of mites, hundreds of which may be found in a single cyst. When many such cysts are present upon the skin the birds quickly grow thin and perish.
2. The **worm-shaped pigeon-mite** (*hypodectes columbarum*) is found in the subcutaneous cellular tissue, upon the serous membranes of the abdominal cavity, and also in the connective tissue of the aorta, and represents the larva or chrysalis (nymph) of *falciger rostratus*. It hinders the processes of moulting and feather production, and also causes slight inflammation.
3. The **Feather-quill Mite of Fowls and Pigeons** (*syringophilus bipectinatus*) lives in the quills of the tail and wing-feathers, and causes deformity in the quills, loss of feathers and irregular moulting. The feathers have a sickly, dull, lustreless appearance, and are usually bent at the point of their pivot ; the quills lose their transparency, and become filled with a dirty, yellowish-grey powder. The mites do not seem to cause any further injury. Heller makes a statement that in Schleswig-Holstein from 70—90 per cent. of all fowls are infested with *syringophilus bipectinatus*. On the peacock we find *syringophilus uncinatus*.
4. The **Feather-mite of Pigeons** (Zürn) lives in great numbers among the feathers, and may give rise to emaciation and even fatal debility. Zürn presumes that the so-called scabies of pigeons on the front part of the head is caused by this mite. (Apparently it belongs to the mallophagi.)

5. The **Analges Minor** discovered inside the quills of fowls by Nörner is perfectly harmless, effecting no change in the quills whatever, and is therefore more of scientific than of practical interest.

6. The **Common Bird-mite** (*dermanyssus avium*) must be reckoned as a blood-sucking parasite, and one of the most troublesome and dangerous of all for poultry. It occurs mostly on fowls and pigeons and also on cage-birds, which it generally attacks at night. But it also lodges on horses, dogs, cattle, cats, and even on man. Its abode during the daytime is in crevices of the floor, cracks in the walls or cages, and also on the under side of the perches. The unrest caused among poultry by these nightly visitors is considerable. Indeed, young ones may even perish from loss of blood, and the older ones grow anaemic and thin. Zürn found that the so-called "greedy sickness" or bulimia of canaries is due to the presence of enormous numbers of bird-mites. They are also often found in the nostrils, where they produce catarrh of the mucous membrane, from which younger birds may die; also in the auditory passages.

7. The **Autumn Grass-mite** (*leptus autumnalis*) is also found on fowls. It bores into the skin at the root of the feathers, producing red dots and little pustules. In consequence of the intense irritation, epileptiform fits may supervene. Young hens scream, flap their wings, fall to the ground and mostly die in a few days. Older ones perish of anaemia.

8. The **Tick of Pigeons** (*argas marginatus s. reflexus*) plagues the pigeons of France and Italy by sucking blood in the night, and may even kill them. It produces a toxic albumen, which has some resemblance to the poison of the viper and puff-adder. Sometimes it also bites men.

9. **Feather-lice** are wingless mallophagi (fur-eaters) and correspond to the hair lice of mammalia. They feed exclusively on feathers and epidermal-scales, but can attach themselves to the skin by their eating apparatus. For details of their families and species, see Zürn's "The Diseases of Domestic Poultry." In large numbers, these creatures can cause disturbance of nutrition in their hosts; and they multiply fastest on sick birds.

10. The **Bird-flea** (*pulex avium*) specially infests pigeons.

II. **Bugs** (*acanthia lectaria*) may spread themselves from beds to poultry and pigeons (Lucet).

12. Finally, the **larvae of beetles** (*tenebrio molitor*, *dermestes lardarius*, *necrophorus*, *silva*) are said to attack pigeons, and even inflict upon them fatal injuries (Kitt.)

For the extirpation of these numerous parasites many remedies are recommended. According to Zürn, the etherial oils (aniseed, rosemary) diluted with 1 : 20—50 of water or oil are good against feather-quill mites and feather-mites (Nos. 3 and 4 above); the same, too, with Peruvian balsam and styrax (1 : 4 spiritus), combined with great cleanliness and an abundance of sand. Sprinkling the plumage with  $\frac{1}{2}$ —1 per cent. creolin-water is a very excellent plan. Against the bird-mite (No. 6) we use Persian insect-powder, or blow sublimated sulphur on to the feathers, or wash them with dilute etherial oil of aniseed. Moreover, the cracks and crevices of the wood-work, perches, etc., must be smeared on a morning with creolin, carbolic acid or petroleum, and the shed if possible swilled with hot water, to which a small quantity of the etherial oil of aniseed has been added. When the fowl-house is of brick, its walls should often be whitewashed, and bird-cages scalded. For feather-lice we also commend Persian insect-powder, or any etherial oil; also decoctions of aniseed, parsley-seed, etc. The birds should also have abundant opportunity to bathe themselves in sand, which enables them to get rid of the parasites. The sand is best mixed with ashes, which may with advantage be scattered alone upon the floor of the fowl-house. Finally, it is recommended that the roosting perches be isolated by oil-cups fixed upon the bearers which carry their ends. It is also said that by hanging a pocket-handkerchief over the bird-cage at dusk and removal of the same at daybreak the mites may be caught and destroyed.

## CHAPTER XI.

### DISEASES OF THE LOCOMOTORY ORGANS.

#### MUSCULAR RHEUMATISM.

**Notes on Rheumatism in General.**—Under the name of "Rheumatism" have long been classed all those diseases of the locomotory organs which arise from cold, and run their course with signs of severe pain, often wandering strangely about the body, and frequently producing much incapacity for movement in the organs affected. Among these organs special mention must be made of the muscles, aponeuroses, tendons and tendon-sheaths, of the periosteum and also the articular synovial membranes. But the connection of all these "*rheumatic*" affections is obviously doubtful; and we might with equal justice include all other complaints which arise from cold under the same name, such as rheumatic peritonitis, pleuritis or colic. But an even stronger reason for rejecting this classification lies in the fact that, according to more recent investigations, cold cannot be accepted as the exclusive source of these "*rheumatic*" diseases of the locomotory organs; for some of them are probably of infectious origin. It is, therefore, advisable to divide them, and to consider as rheumatic that form which is really caused by cold, viz., muscular rheumatism, leaving for separate treatment the articular form, which is evidently an infective condition. But we must admit that in our domestic animals a differentiation between muscular and articular rheumatism is not so easy as in man, and in many cases is impossible. The diagnosis of muscular rheumatism in particular is often rendered very difficult by the absence of all subjective symptoms; and, moreover, complications of the two forms are much commoner in domestic animals than in man. Besides the difference of etiological relation, the difference of course also demands a separate description. Muscular rheumatism must be regarded

as muscular inflammation (*myositis rheumatica*), whereas articular rheumatism represents an inflammation of the joints (*polyarthritis serosa*). The two conditions have nothing in common beyond their accompanying motor disturbances and pain.

**Etiology.**—Muscular rheumatism, or rheumatic muscular inflammation, occurs oftenest in horses, dogs and cattle, but is not rare in sheep and pigs. Of the dogs treated in the Berlin dog-hospital, just over two per cent. suffer from this complaint. Cold is universally regarded as its chief cause. In the first place, we put damp, cold weather, cold winds and draughts, then badly kept stables, wet pastures and rapid cooling of the body after violent exertion, etc. For instance, in the breeding establishment at Mezöhegyes acute muscular rheumatism appears almost as an epidemic among the foals of from six months to two years (Tatray) in spring and autumn, and as the result of cold. Cold baths are in sheep a cause of muscular rheumatism, as well as lying out of doors on very cold nights; and in dogs a cold sleeping-place or fetching objects out of very chilly water; and consequently the disease is much more common in the colder seasons. Among very pampered or young animals we find a predisposition, and also among tender merino lambs, or animals that have long been kept in warm stables, or been too highly fed or too little exercised. For the latter reason the ailment is common among fat dogs. Animals once attacked are more liable to a recurrence of the trouble. Exactly how muscular rheumatism is caused by cold is not yet satisfactorily explained. Probably the irritation produced by the cold upon the temperature nerves of the skin is by them reflexly transmitted to the nerves and vessels of the muscles, causing first of all disturbances of circulation and changes in the composition of the individual muscular fibres, which finally assume the character of a decided muscular inflammation. This explanation is based upon the physiological fact that, even under normal conditions, the metabolism in the muscles is stimulated by the action of a lower temperature upon the skin.

**Older Theories.**—The earlier opinion that the effect of chill was to suppress perspiration and thereby throw back certain injurious substances into the blood, and in consequence of this blood-dyscrasia set up inflammatory changes in the blood, can no longer be accepted. Neither does the hypothesis of an increased generation of lactic acid

in the muscles, as a connective link between the chill and the muscular affection, appear to be justified; for neither by artificial suppression of the activity of the skin (varnish) nor by the introduction of lactic acid into the body, has anyone yet been able to produce rheumatism. The conception also of muscular rheumatism as a purely neuro-pathological process or muscular neuralgia is contradicted by the proof of inflammatory changes in the muscles themselves. Nevertheless, it must be admitted that under the name and aspect of muscular rheumatism several other disturbances—neuralgic, traumatic and circulatory—have often been included. Evidently a muscular inflammation exactly resembling, clinically, a rheumatic myositis can also be produced by over-exertion; but such traumatic muscular inflammation must not be confounded with muscular rheumatism, although it is by no means always easy to differentiate between them. Leube has recently declared that muscular rheumatism is an infective malady.

**Post-mortem Lesions.**—By no means uncommonly we discover no pathological change in the muscles attacked by rheumatism; but this is only in slighter cases, in which the hyperæmia or slight inflammation present during life is imperceptible after death (like the disappearance after death of milder catarrhal affections upon mucous membranes). When the attack is more severe, the well-marked signs of muscular inflammation are all present: hyperæmia, haemorrhage, serous exudation into the interstitial connective tissue (perimysium internum), softening, discoloration and disintegration of the muscular fibres. Siedamgrotzky found in all the aponeuroses of a dog a diffuse redness and serous infiltration, the muscular apparatus being bright red and the capillaries injected; and in another dog, suffering from chronic rheumatism of the masticatory muscles, an intense interstitial chronic myositis in all these muscles. Brückmüller also saw the inter-muscular connective tissue infiltrated with serum and charged with a reddish yellow, gelatinous fluid, the muscular fibres, which were very pale, had lost their transverse striation and undergone fatty degeneration. If the rheumatic processes be long continued, inflammatory proliferation of connective tissue may set up in the muscles, producing the so-called rheumatic induration, i.e., a deposit of connective-tissue bands; and finally, an extreme muscular atrophy sometimes appears in the affected muscles.

**Symptoms.—A. In Horses.** The phenomena of muscular rheumatism in horses are usually restricted to single groups of muscles, particularly those of the extremities; only rarely does the condition attack the entire muscular apparatus of the body. The animal is attacked suddenly, displays a stiff,

strained posture and action of the parts affected, viz., the extremities; can only with difficulty be made to stand up or move backwards, makes very short steps, not bending its joints as it does so, but keeping them as straight as possible. Such movements are often accompanied by a peculiar, loud cracking of the joints. The affected muscles are painful and hard to the touch; and sometimes their entire surroundings are in a state of œdematosus swelling.

A characteristic sign is the shifting (metastasis) of the rheumatism from one part of the body to another, e.g., from a fore leg to a hind leg, when the limb first attacked may very quickly recover. Also the marked liability to a recurrence of the disease, and its improvement or even complete disappearance after prolonged exercise are further characteristics. The muscles most frequently attacked are those of the shoulders (*omodynia rheumatica*) and of the loins (*lumbago*). When the shoulder is affected its power of movement is hindered, especially in raising and advancing the limb, the stride is shortened and the horse goes "shoulder-lame." On backing, the foot is dragged and lameness is increased on going uphill or on soft ground. When the extensor muscles are particularly affected, the unfitness is marked on turning quickly. Lumbago shows itself in a stiffer carriage, sensitiveness and weakness in the lumbar region, a dragging gait, difficulty in getting up, and even in complete inability of the hind legs, so that the hind quarters sink and the horse cannot rise again. The muscles around the hip-joint may also be implicated (*hip-lameness*), when a dragging gait with diminished flexion and forward movement of the affected limb are observed, and even a collapse if more weight be thrown on that side than on the other. It is not easy to decide, in the absence of all subjective symptoms, whether rheumatism of the inter-costal muscles (*pleurodynia rheumatica*) ever occurs in horses as in men. Along with these local phenomena we observe some degree of fever when the muscular rheumatism is widespread (we have noted 104° Fahr.), also increased rapidity of pulse and respiration; but, on the contrary, when the rheumatism is local there is no fever, and only a slight increase in the pulse-beats. Severe cases of muscular rheumatism are sometimes complicated with other disorders arising from chill, such as catarrh of the breathing and digestive apparatus, with colic, pleuritis, affections of the tendons and grease; and also, though only rarely, with articular rheumatism. In this manner the aspect

of the disease is naturally much altered and may even take a fatal turn.

The *course* is very often rapid, in which case it is often marked by a sudden disappearance of the gravest symptoms and a somewhat brief duration of the whole illness. Recovery then occurs in a few days or, at most, a week. There is, however, always a tendency to relapse. But muscular rheumatism often takes a chronic course and may become extremely obstinate and drag on for weeks and months. This is especially the case when the shoulders or loins are attacked. In rare cases of severe and general rheumatism death may result.

**B. In Cattle** muscular rheumatism makes its appearance very much in the way described in horses, only that complication with articular rheumatism is more frequent. The favourite seats are, the shoulders and lumbar region. It may be localised or almost every muscle of the body may be implicated, so that the beasts stand quite stiffly, unable to stir either neck or limbs and groaning piteously. Sometimes the appearances are like those of epizoötic cerebro-spinal meningitis. The affected muscles feel tense and painful to the touch. In very acute cases the internal temperature is raised ( $104\text{--}106^{\circ}$  Fahr.), and the breathing is occasionally extremely laboured. As regards the course of muscular rheumatism we must observe that in cattle it frequently becomes chronic; but severe acute attacks may rapidly lead to death.

**C. In Dogs** the rheumatic process is usually localised in the muscles of the neck, shoulders and back, but not unfrequently affects the whole body. A prominent symptom is the animal's crying; many howl on the least attempt at movement or on the gentlest touch, others even from fear of being touched. That which gives a dog most pain is to lift it from the ground, elevate its head, stretch out its limbs or attempt palpation of the psoas muscles (*lumbago rheumatica*). The animal's movements become stiff, strained, timid, its neck is often held with peculiar stiffness, and the patient is frequently quite unable to move forwards, but lies down continuously. The ascent of stairs is particularly difficult and often quite impossible. But the sensitiveness of the separate muscular groups becomes very quickly modified, and those which have seemed most severely crippled may often in a very short time be fit for use again, while the rheumatic condition has meanwhile

settled elsewhere. The dog's appetite is often quite normal even during very severe attacks, but mastication is naturally impeded if the jaw muscles be among those attacked. Constipation is also often observed, as the attitude for evacuation has become painful and is therefore avoided when possible. Muscular rheumatism in dogs not seldom takes a chronic course, and these inveterate cases sometimes defy the most persevering treatment for months. During their course muscular atrophy sometimes develops.

D. **Among Sheep** it is usually lambs which are attacked by muscular rheumatism, and the condition may be either local or general. The animals walk with a stiff gait, as though upon stilts, hold both back and neck stiffly, the head and neck are often persistently bent to one side, and there is frequently lumbago. The affected muscles are painful and hard to the touch; movement is avoided and the lambs remain lying. The course is generally rapid, and cure may take place in four to six days, but in other cases the disease becomes chronic.

E. **In Pigs**, as in cattle, this form of rheumatism is often complicated with the articular variety. The animals have stiff legs and walk stiffly and with pain; the back also is stiff. Gastric disturbances are also said to be an accompaniment in pigs, and a weakness of the hinder parts resembling paralysis often remains behind.

**Differential Diagnosis.**—Muscular rheumatism may be confounded with various surgical affections of the extremities, particularly those of the hoofs, tendons, bones and joints. (See surgical text-books on this subject.) From articular rheumatism it may with ease be so far distinguished as that this is always accompanied by swelling, raised temperature and painfulness of the joints; and yet in articular rheumatism the inflammation of a deep-seated joint which cannot be reached for palpation may always present the aspect of a muscular rheumatism; in which case we must endeavour to find out if any of the muscles be affected and painful when manipulated. Much more difficult is the differentiation of a rheumatic lumbago from a paralysis of the hind-quarters caused by spinal affections or external traumatic influences. Moreover, rheumatic lumbago in horses, as seen in the course of acute muscular rheumatism, greatly resembles the phenomena of the paralysis of the hind-

quarters which appears in haemoglobinæmia, which must also be regarded as a rheumatic myositis. But in ordinary muscular rheumatism we have no haemoglobinæmia or haemoglobinuria. Rheumatic haemoglobinæmia and muscular rheumatism in horses are therefore ailments only different in their intensity. Finally, muscular rheumatism may be confused with pleuritis, nephritis, osteo-myelitis and certain rheumatoid affections, such, for instance, as appear in the course of many infectious diseases, say in cases of bronchiectases. In cattle the malady is sometimes mistaken for cerebro-spinal meningitis.

**Therapeutics.**—The treatment of localised muscular rheumatism is chiefly external. It consists in rubbing the affected parts, after previously sprinkling with irritant remedies, e.g., with spirits of camphor, either alone or with oil of turpentine or of rosemary (1 : 10), with etherial mustard oil (1 : 50), spirits of ammonia (1 : 15), tincture of arnica, etc. In this operation, friction and not the drug plays the chief part; we may also use Priessnitz's bandages, or cold douches, followed by warm packing, or massage, and, finally, try to excite local perspiration. To this latter end let the affected parts be washed with soapsuds as warm as possible, vigorously rubbed and then warmly wrapped up. In chronic and very obstinate rheumatism of the shoulder, a subcutaneous injection of veratrine is highly recommended. For a horse inject it beneath the skin of the shoulder in doses of  $\frac{1}{2}$ - $1\frac{1}{2}$  grains in 15-30 minims of alcohol, increasing the dose daily by about one-sixth of a grain, and omitting the injection every fourth or fifth day. After each such operation it is well to exercise the horse, owing to the signs of restlessness which immediately follow it.

In cases of general muscular rheumatism the internal administration of salicylic acid or salicylate of soda is most to be commended. To larger animals (horse, ox) we give both remedies in doses of  $\frac{1}{2}$ - $1\frac{1}{2}$  oz. thrice daily, and to smaller animals (dog, pig, sheep) 30 to 120 grains a day. As a rule, salicylate of soda is to be preferred to the acid, which, having a slightly corrosive action on the mucous membranes, is apt to interfere with appetite. This salt is usually given to large animals in pills or electuaries, to smaller ones in solution. An ounce may be given to the horse in the form of an electuary every three hours until three or four doses have been given. In the case of the dog the solution may be repeated hourly until acute symptoms have disappeared. Salol is also used in place of the

two remedies just named. It is said to be preferable to salicylic acid, and that it leaves no bad after-effects and does not irritate the stomach; it seems moreover to act in the body like disguised salicylic acid. Administer salol to dogs in doses of 4-16 grains and up to 16 grains to 1 dram per diem, but for the larger domestic animals it is generally too expensive. We can confirm its good effects from personal experience. Along with salol, antipyrine is a very good remedy for rheumatism in dogs, and we prescribe it for them in the following form: Rx. antipyrin, 1½ drams; aq. destill., 5 oz., a tablespoonful every three hours. Salipyrine has a similar effect.

The administration of laxatives is also very common, and tartar emetic especially is given to horses in feverish, acute muscular rheumatism. For dogs tincture of colchicum has been recommended as a gentle purgative and rheumatic specific —5 to 15 drops twice or thrice daily. A general perspiration may also be provoked partly by external means (wrapping the whole body in wet clothes and then surrounding it with woollen rugs), and partly by diaphoretics internally administered. Of these, though, the only ones which act upon horses are arecoline hydrobromide (1½ grains) and pilocarpine hydrochloride in very large doses (full-grown horses 6-12 grains; foals 3-5 grains, dissolved in 1-2½ drams of water), and injected beneath the skin. Hübner thus used pilocarpine successfully on a nine-months foal against muscular rheumatism (4 grains in 1 dram of water). Sweating did not break out for two hours, but lasted then for three hours. Siedamgrotzky, on the contrary, had no success with three horses after 4-5 days' continued injection of 3-6 grains of pilocarpine. Of course, all chill must be carefully warded off from animals thus freely sweating. They must also have rest, be kept warm and be moderately dieted.

## ARTICULAR RHEUMATISM.

**Definition.**—Articular rheumatism is an infective febrile disease accompanied by inflammation of several joints, and general systemic disturbance, it is commonest among cattle, rarer in other animals, but is found in goats, dogs and pigs, and very rarely in horses. Out of 70,000 dogs treated in Berlin, there were only 92 cases of articular as against 1,462 of muscular rheumatism. Only very few of the arthritic affections of sucklings belong to this category. It is, therefore,

best to treat them along with pyæmic articular inflammation, with which they have often been confused.

**Etiology.**—The main source of articular rheumatism was formerly sought in colds, for the production of which the same factors were blamed (draughts, damp, cold, bad quarters) as in the muscular form. But there is no doubt that, as in man, so also in our domestic animals, articular rheumatism is an infective disease in which chill only acts the part of a predisposing cause. Proofs of its infectious nature are : The general feverish signs, as well as the initial stage characteristic of all infectious maladies, the simultaneous affection of several joints, often far removed from each other ; the endocarditis which often also appears ; its outbreak in model farms and byres, where taking cold is carefully guarded against ; and the observation made by many authorities that articular rheumatism is indisputably connected with the retention and putrefaction of the after-birth. Auer noticed the disease almost exclusively in younger cows after their second or third calf, and in two-thirds of all the cases observed (67 in all) could prove abortion or retention of the after-birth. He therefore regards the resorption of septic matter in the uterus as the direct cause of articular rheumatism. Dinter and Dessart also saw the disease exclusively in cows that had newly calved. We may therefore include that in cattle the puerperal uterus is the chief source of the infectious matter which produces this disease. Barthélemy observed an epizoötic articular rheumatism in goats. As in other infective diseases, chill favours the penetration into the body of the infective matter and also its multiplication, by rendering it weaker and less able to resist. But the infective substance in question is not yet bacteriologically known. It is not impossible that various bacteria may produce the aspect of articular rheumatism, and that, therefore, the affection does not in this respect represent an etiological unity.

In man a *plasmodium* penetrating from the tonsils has been suspected to be the infectious substance, as articular rheumatism is frequently complicated with tonsilitis (according to Fiedler 158 times out of 655 cases). Edlefsen maintains that it is a miasmatic contagious disease which occurs most frequently when there is a decrease in the atmospheric pressure, and may be confined to certain localities ("rheumatic houses"). Secondary or predisposing causes are over-exertion and chills.

**Post-mortem Lesions.**—The anatomical changes in

the joints consist mainly in a serous synovitis and, in chronic cases, in inflammatory, degenerative modifications of the joints; a suppurative synovitis is only very rarely observed. Generally, several joints are affected at the same time. When the course is acute, we find the synovial membrane highly reddened, even permeated by haemorrhagic areas, opaque and swollen, sometimes also thickened, and the synovial villi vascular and enlarged. The synovial fluid is increased, coloured red and often also opaque. The articular cartilages are at first reddened, later they turn yellow and display a rough, velvety surface. The tissues round about the joints also appear to be injected by haemorrhages and serously infiltrated (peri-arthritis); and the connective tissue especially is swollen and gelatinous, the neighbouring muscles are oedematous and softened. The articular ends of the bones are, moreover, hyperæmic and even haemorrhagically infiltrated, and likewise their medulla. During the chronic course of the disease the synovial membrane becomes very much thickened, and the inner surface of the joint often contains a coating of connective tissue very rich in cells (arthritis pannosa); the articular cartilages undergo fatty-disintegration, show an ulcerative loss of substance and are partially detached. In rare cases, an arthritis deformans is developed, as in man. Thus Moore noticed the bones of the root of the tarsus become ankylosed to the metatarsus in a dog suffering from chronic articular rheumatism, and also irregular formation of osteophytes. Among general physical changes we may mention that endocarditis, pleuritis and peritonitis sometimes occur; other changes are of no moment.

**Symptoms.**—The most important of these is swelling of the joints. This often appears quite suddenly, frequently overnight and may soon attain considerable dimensions. It generally attacks several joints on the same or on different legs, and seems to prefer the carpal, tarsal and knee-joints. The other joints are often affected in a distinctly ascending scale. Only rarely is a single joint attacked (mono-articular form). The swellings are hot and very painful, feel tightly stretched and in some spots fluctuating, and extend all around the joint. The adjoining parts (tendons, tendon-sheaths, muscles) are also implicated in the inflammatory process and are swollen. The animals consequently limp so much, that a non-professional observer might suspect a broken bone. The

swellings may be so painful when touched for examination, or when the joints are bent, stretched or turned, that the animals can only with difficulty retain their feet. They therefore avoid every movement or even putting the affected leg to the ground, and often lie down continuously, groan and grind their teeth, and can only with difficulty be made to stand up, and sometimes not at all. Their internal temperature is also at first raised and is on an average  $1\frac{1}{2}^{\circ}$  to  $3\frac{1}{2}^{\circ}$  Fahr. higher; the pulse is small and hard, and, in cattle, 70 to 90 beats a minute. Both appetite and rumination are suppressed, the muzzle is dry, defecation delayed and the secretion of milk much decreased, the milk itself being of a sour flavour and readily coagulating. In addition we may note a striking loss of flesh in cattle, even after the first two days.

In addition to these customary symptoms we in many cases note peculiar complications. First, and especially in cattle, an acute endocarditis may develop, which sometimes leads to sudden death. Then appearances also of pleuritis, peritonitis, pericarditis and laryngitis may be observed, as well as inflammation of the tendon-sheaths. These complications, as also the exanthemata (urticaria) sometimes seen in man, cannot be understood except on the hypothesis of a general infectious disease.

The *course* is usually chronic, seldom acute. In solitary cases a retrogression of the inflammatory articular swellings sets in along with abatement of the fever, and a decided recovery takes place in two to three weeks; but such a benign course of the malady is rare. Very often the cure is only apparent, and a relapse soon occurs. For instance, a severely affected joint may recover with surprising quickness in a few days, while another and distant one may be freshly attacked (migration of articular rheumatism). In by far the majority of cases the course is very wearisome and also unfavourable. The feverish signs disappear, it is true, but the disabilities and lameness remain in the joints. Chronic degenerative changes gradually develop in the latter, the animals go back in condition, yield less and less milk, suffer from digestive disorders and gastro-intestinal catarrh with very violent diarrhoea and grow by degrees weaker and thinner. The groups of muscles above the affected joints (those of the shoulder, etc.), are atrophied, the extremities become stiff and crooked, and the joints remain thickened. Matters may continue thus for two to three months or longer. Dammann noted a case of a horse which lasted

eight months without cure. If the animals be not soon slaughtered, they often perish of exhaustion, their bodies being covered with sores brought on by prolonged lying.

As regards *differential diagnosis*, traumatic and pyæmic arthritis must be taken into consideration, also meningitis (when the vertebral articulations are affected).

**Therapeutics.**—So long as the nature of the infective matter is not more fully known, we can only recommend by way of prophylaxis the avoidance of the most important contributory cause of articular rheumatism, viz., chills. Horses especially must be rubbed down, clothed or walked about after violent exercise until dry. In cattle an early removal of the after-birth and thorough disinfection of the uterus must in all cases be remembered. Internally salicylic acid and salicylate of soda are specific and admirable remedies. For doses and method of administration, see the Treatment of Muscular Rheumatism (daily dose of salicylate of soda for cattle and horses 3 to  $4\frac{1}{2}$  oz.; for dogs and pigs  $\frac{1}{2}$  to 2 drams). Instead of salicylic acid one may give salol, salipyrin, salophen, antipyrin, phenacetin, antifebrin and similar new remedies. By far the cheapest among these is antifebrin. To promote absorption of the arthritic exudations and elimination of the infectious matter through the mucous membranes of the intestines, laxatives may also be administered, especially tartar emetic and large doses of neutral salts. Iodide of potassium may also be tried as a re-absorbent. Among *external means* we suggest warm wrappings about the affected joint, after first rubbing it with an ointment of grey mercury, or of carbolic acid, iodoform, chloroform or camphor. In chronic cases with delayed resorption, one may have recourse to sharp inunctions of the joint (tincture of iodine, cantharides ointment, iodide of mercury). Food must be carefully regulated. Avoid movement of the animal, but keep its stable warm and sufficiently ventilated.

#### PYÆMIC AND SEPTIC ARTHRITIS ("LIMPING") OF SUCKLINGS.

**General Remarks on the Term "Limping" (*Lähme*).**—Under the name of "Limping" a series of ailments in sucking animals (foals, calves, lambs, sucking pigs and puppies) has long been included, which, although essentially differing in cause and nature, are yet alike in manifesting themselves by

serious disturbances of locomotion. The term limping is therefore only a generic name, which should no longer be retained, but replaced, if possible, by more exact scientific nomenclature. Thanks to Bollinger, we can now differentiate better the various diseases so designated, and would therefore indicate the following :

1. *Pyæmic or septic articular inflammation* which develops by metastasis from a septic infection of the navel-wound. This septic-pyæmic polyarthritis is, according to Bollinger, the most serious and frequent cause of the limping of sucklings.
2. *Fatty degeneration of the body muscles*, especially sucking pigs.
3. *Acute articular rheumatism*.
4. *Acute and chronic muscular rheumatism*.
5. *Rickets*, particularly in sucking pigs.
6. *Tetanus and cerebro-spinal meningitis*, especially among lambs.
7. *Tabes*, a disease of foals, which is probably identical with tuberculosis of the intestinal and mesenteric glands.
8. Various other diseases to which sucklings are liable, e.g., intestinal catarrh, broncho-pneumonia, pleuro-pneumonia, general debility, weakness in the extensor muscles of the extremities with consecutive contraction of the flexors (in foals and dogs), etc.

**Etiology of Pyæmic Arthritis.**—The causes of this form of inflammation, which occurs especially among foals and calves and also among lambs, must be sought in a purulent, ichorous inflammation of the navel and its surroundings, with thrombosis of the umbilical vein, suppurative disintegration of the thrombus and distribution of its waste matter by the blood. The starting-point is an infection of the navel-wound by dirt from the floor of the stable. Uffreduzzi cultivated two separate bacteria—a micrococcus and a bacillus—from the bodies of calves which had died of this disease, and successfully inoculated therewith, when he observed not only pyæmic, but also septicaemic symptoms, so that it may be presumed that from the infected navel-wound both pyæmia and septicaemia may develop. Buch found in the liver micrococci which had a likeness to the diplococci of pleuro-pneumonia. Turner found the same bacteria in the membranes of the ovum of brood-mares suffering from epizoötic abortion, as in the organs of foals that had died of "limping." Cultures of the same

injected into the vagina of pregnant mares produced abortion, with arthritis in the aborted foals.

The cause is to be found in connection with the very insufficient care which is bestowed upon the navels of our domestic animals. Bollinger affirms that a thick, gelatinous character of the umbilical cord predisposes to this affection; the same, too, may be said of wrenching the umbilical vessels, or tearing them off too short, contusion of the wound, or injury to the abdominal walls. According to Gmelin, the predisposition must be sought in the navel itself. He states that necrotic processes take place quite normally within the umbilical ring; that, moreover, the umbilical arteries become plugged by thrombi, the central ends of which are connected with the circulating blood and the peripheral ends with the outer air and its neighbouring necro-biotic processes; so that the peritoneum which covers the inner navel-ring is drawn into direct sympathetic affection with all the morbid processes going on within the umbilicus. Finally, too early births in winter time, necessitating a long sojourn in the stall, greatly increase the risk of infection from the manure-soaked ground.

The disease sometimes appears enzootically in herds and breeding stables. According to Hering, of 187 sucking foals which died during 15 years in the provincial breeding stud at Marbach in Württemberg, 85 perished of "limping."

The question of *hereditary transmission* of "limping" was in former times universally answered in the affirmative. The contrary opinion was first energetically proclaimed by Hering in 1863. But even quite recently professional men have maintained that an intra-uterine infection of the foetus by the mother animal may occur, in addition to any subsequent affection of the navel, because the navel of foals suffering from "limping" very often shows no morbid signs. But this argument does not seem to us to be valid, on the one hand, the navel may remain apparently sound in spite of infection in the body, an analogy to which fact may be found in the womb during paresis of parturition, and on the other hand, the septic navel-affection may heal, while the pyæmic processes go forward in the body. How else can we explain the undoubtedly favourable results of prophylactic care of the navel (Bollinger, Gmelin), if there be an intra-uterine, haematogenous infection? The possibility of such intra-uterine infection does not seem to us to be positively proved, yet it may easily happen that some foals bring a certain predisposition to pyæmic polyarthritis with them into the world, in so far as they are burdened with a low vitality and feeble constitution, qualities which may be inherited from their mothers.

**Post-mortem Lesions.**—The phenomena revealed are essentially those of suppurative polyarthritis, along with

other changes characteristic of the pyæmic process (pyæmic form); in other cases the aspect of septicaemia is more pronounced (septic form). The navel-wound, if not already healed, shows signs of inflammation and suppuration, the umbilical cord and surrounding region are swollen, the edges of the navel-ring are ulcerated, within the inner umbilical ring abscess-formation is found, and through the opening of the navel drops of pus can be squeezed. Alongside this omphalitis—which, as stated, may have healed and left no external morbid signs—we find, on going deeper, a suppurative thrombo-phlebitis and thrombo-arteritis umbilicalis, with thrombosis of the portal vein and its branches in the liver. In the joints the synovial membrane is injected, swollen and thickened, the synovial fluid is opaque, increased in quantity, mixed with flaky coagulae and later of purply purulent character (empyema of the joint). The articular cartilage is ulcerated or destroyed, sometimes even the bony tissue is necrotic and the articular capsules perforated. Abscesses form round about the joints between the tendons and muscles, the latter being purulently infiltrated. The muscles over the affected joints have undergone fatty degeneration. The most marked lesions are found in the knee and hock joints and the joints attaching the head to the neck. Dissection also reveals metastatic foci in almost all organs, especially in the liver, lungs, brain, kidneys, muscles and subcutaneous cellular tissue. In the liver these foci range in size from a millet-seed to a cherry or larger; and in the lungs from a millet seed to a hen's egg. They are at first dark red and later turn yellow (so-called suppuration of the lungs). Pleuritis, endocarditis and pericarditis are also observed, as well as bronchopneumonia, inflammation of the tendon-sheaths, peritonitis, cystitis, suppurative iritis, ophthalmia, meningitis, etc. Finally, the heart, liver, kidneys and muscles are in a state of fatty degeneration.

**Symptoms.**—Pyæmic arthritis in the greater number of cases develops in the first days or weeks after birth. Of 67 foals, says Hering, 40 (or 70 per cent.) sickened and died in the first three weeks of life. Bollinger, taking his figures from another breeding establishment, says 75 per cent. Usually a few general symptoms precede the appearance of the arthritic affection, such as fever, diminished or suppressed desire to suck, etc.; but these may be absent. The swellings on the joints generally develop very quickly. The knee and hock

joints, as well as the elbow, hip and shoulder are most frequently attacked; but also the fetlock and coronet, while even the joints of the spine and ribs do not escape. The swelling is hot and painful, very tense, and at first often disappears rather quickly. But in its later course it has a tendency to form abscesses, the pus within the joint opening itself a way outwards. Generally several or even all the joints of the body are inflamed at once. The animals grow very lame, are afraid to touch the ground with the affected legs and lie down a great deal. To these symptoms diarrhoea is soon added, the faeces being greyish-white and bad-smelling, and with the diarrhoea constipation alternates. The animals grow very thin, weak and debilitated. Furthermore, according to the position of the metastasis, various complications may be observed (inflammation of the lungs, sudden swellings in different parts of the body, cerebral phenomena, blindness, etc.).

The *course* is generally acute in foals; the average duration of the illness being two to three weeks, but a fatal end may occur even in a few days. According to Gmelin's observations, the foals which died of pyæmia attained an average age of 26 days; the disease declaring itself on their 17th day and killing them after an average duration of nine days. The *prognosis* is very unfavourable, estimates of mortality ranging from over 50 per cent. to 90 per cent. of those afflicted. The remainder only recovered very slowly, continued for a long time to be sickly and were often backward in growth. The swellings on the joints and the stiffness which they cause are also long in passing away, or even become permanent.

In *calves* the prognosis seems to be more favourable, the mortality, according to Strebcl, being only 35 per cent. But the course with them is slower, sometimes even chronic, and the pyæmic affection is less widely diffused through the body. Sometimes only one joint, that of the patella, is affected; and this was noted, according to Strebcl, in older animals (of three to five months). Probably this more favourable course in calves is due to the circumstance that, as compared with foals, their umbilical arteries tear within the abdominal cavity and can therefore retract further, whereby their thrombi are better protected from infection (Gmelin).

**Differential Diagnosis.**—In order to distinguish pyæmic arthritis from acute articular rheumatism—two complaints very easily confounded—the following points may be

of importance. First of all, the development of a disease in the joints of sucklings a few days or weeks old proclaims pyæmic arthritis, especially if a suppurative inflammation of the navel can at the same time be detected. Moreover, the formation of abscesses in articular rheumatism is so rare an occurrence, that it may always be taken as a sign of the disease under consideration. Finally, articular rheumatism seems to be much less common among sucklings.

**Therapeutics.**—In accordance with the pathogenesis of the ailment, its treatment is especially prophylactic. Bollinger recommends above all things strict attention to the navel, careful ligature of the umbilical cord, antiseptic treatment of the navel-wound, with a protective bandage round the belly; or, if necessary, the latter may be replaced by daubing with tar. Gmelin advises, after ligaturing the umbilical cord, painting the cut surface with concentrated carbolic acid until (four to five days) the rest of the cord assumes a parchment-like appearance. Then clip off the navel-stump with scissors and paint the wound daily (seven to eight days) with sublimate gelatine (1 per cent.) until it heals. After introducing this plan the disease completely vanished from a stud where it was formerly very prevalent. The stalls must also be kept very clean, dung and urine be removed, the floor disinfected and good, dry straw provided. In the disease itself antiseptic treatment of the umbilical region must be adopted (cleansing with creolin or carbolic solutions, application of a 10 per cent. ointment of creolin, carbolic or iodoform). Also anti-febrile and anti-symptomatic remedies must be used, especially camphor (subcutaneous injection of spirits of camphor in doses of 20 to 80 min.); also stimulants (alcohol, wine). The treatment of the affected joint is purely surgical. Above all, the pus must be removed as soon as possible by puncture or incision.

**Fatty Degeneration of the Muscles in Sucklings.**—Under this name an affection of the locomotory organs has been described, which occurs chiefly among English pigs and fine-bred lambs, more rarely among foals and calves. It is, however, only a partial symptom of a general congenital fatty degeneration of the body, and ought rather to be classed under the heading of obesity. It is developed during the second half of the foetal life, and its causes may be sought in an inherited predisposition and in a tendency to fatness in the mother, found especially among breeds which reach maturity early; also in the mother's resting too much in the stall, combined with over-richness of feeding. Fürstenberg also blames breeding from animals too closely related. The anatomical

*changes* consist in general anaemia, fatty degeneration of the body muscles (which often cannot be recognised as such, but shine like lard or look as though cooked), also in fatty degeneration of the liver, kidneys, brain, pancreas and of the glands of the intestinal canal. The *symptoms* reveal themselves in general weakness, dull, sluggish movements and even inability either to walk or stand. In severer stages of the disease the animals lie in complete apathy and often die speedily without any further complications, or after attacks of diarrhoea, spasms, and paralysis. The ultimate cause of death is frequently oedema of the lungs. The *treatment* is only prophylactic. An introduction of fresh blood by frequently crossing the breed, exercise and more restricted feeding of the mothers during pregnancy are the most important matters. Any medicinal treatment of the young animals is fruitless.

**Infectious Articular Inflammation in Young Geese.**—Lucet has observed an articular inflammation in goslings (caused by *staphylococcus pyogenes aureus*) which appears sometimes sporadically and sometimes enzootically. It manifests itself in lameness, diarrhoea, fever, much lying down, swelling of the joints and in death after one to three days. More rarely the course is sub-acute and chronic (formation of ankyloses). The disease can be induced in goslings by inoculation with *staphylococcus pyogenes aureus*.

## OSTEOMALACIA AND RICKETS.

**General Notes on Diseases of the Bones.**—Those diseases of the bones which belong to the realm of internal pathology, of which osteomalacia and rickets are by far the most important, might very well be relegated to the chapter on general disturbance of nutrition. On the other hand, it has long been the custom to rank them among diseases of the locomotor apparatus because, during life, their most prominent symptom has been a defective power of motion. A purely etiological classification is still impossible, as, in spite of numerous experimental investigations, their etiology is not yet sufficiently cleared up. Neither has pathological anatomy hitherto been able to furnish a clear survey of the pathogenesis of these bone diseases. It may perhaps be best to classify the bone diseases of internal pathology into two great groups:—

1. The *bone diseases of the earlier period of life*, of which *rickets* is the most important.
2. The *bone diseases of later life*, among which *osteomalacia* is the most important.

As regards the relation between rickets and osteomalacia opinions by no means agree. But the two diseases are obviously related. This is shown by the fact that at certain times they

appear simultaneously in the same stable or the same herd and that both may be experimentally and simultaneously produced in one and the same animal ; and the prime cause of each is to be sought in a deficient supply of lime. It is true the pathological changes in the bones show great differences, but if we bear in mind that, even normally, there are considerable structural differences between a growing and a fully developed bone, we may assume that the varied pathological aspect of rickets and osteomalacia may arise solely from the different ages of the bones affected. According to this, rickets may be regarded as osteomalacia modified by the condition of growth in younger bones. We therefore define the relation of both as follows :—

1. *Osteomalacia* is a re-softening of the bones of mature animals in consequence of resorption of their lime-salts (halisteresis).
2. *Rickets*, on the contrary, arises from the bones of young animals remaining soft owing to deficient calcification.

**Experimental Investigations into the Etiology of Rickets and Osteomalacia.**—A great many experiments have been made to find out if possible the causes of these two conditions, but the results cannot be considered satisfactory. Nevertheless, they have given considerable help towards an understanding of the diseases, and we must therefore devote a few sentences to their discussion. The following three theories, which are briefly known as the "inanition," the "acid" and the "inflammation theories" have become specially important for pathogenesis.

1. *The Theory of Inanition* postulates as the chief cause of both diseases of the bones a deficiency of lime in the food. Roloff and E. Voit produced decided phenomena of rickets in young dogs and pigs by continuously feeding them on food that was poor in lime, and those of osteomalacia in a full-grown goat and sheep. The former manifested backwardness in the formation of bone, with a lack of firmness and bulk in the several bones with all the other signs of rickets ; swelling of the epiphyses, bending of the ribs, shoulder-blades, etc., crooked limbs, with a heavy, painful gait, to the extent even of inability to use the legs at all.

These experiments were confirmed by others made by Chossat, Milne-Edwards and Lehmann, but are contradicted in their results by those of Weiske, whose animals, fed on food lacking in lime, perished, it is true, but did not become rickety.

But the objection is justly raised to his conclusions, that the animals he experimented on died, not of deficiency in lime, but of inanition, before any changes could be produced in the bones, and the fact that rickets can be artificially caused is by no means disproved. These experiments in feeding of Roloff and Voit have been fully complemented and their result confirmed from clinical practice. Poverty of lime has always been universally regarded as the chief cause both of rickets and osteomalacia, and the fact very often proved. Moreover, osteomalacia is evidently closely connected in pregnant and milk-yielding animals with an increased abstraction of lime from the system. Finally, the fact that both affections can be cured by an increased supply of lime greatly strengthens this view. (See the etiology and therapeutics of the individual maladies.) The theory of lime-inanition may, therefore, be regarded as proved, not only by experiment, but also by the facts of actual practice.

2. *The acid theory* is based on the assumption that during catarrh of the stomach and bowels, which is peculiarly frequent in young animals, an abnormal quantity of lactic acid is generated from the milk and carbo-hydrates absorbed, and that this acid, after having passed into the blood, dissolves out the lime-salts of the bones and causes proliferation of the bony tissue. Heitzmann seemed to prove this theory by producing rickets in animals by persistent subcutaneous injection of lactic acid and feeding them on the same substance. But Roloff, Heiss, Tripier and others failed to confirm his statements by the experiments they also made with lactic acid. Roloff holds that, as Reitzmann avoided at the same time all foods containing lime, he obtained his results not by the supply of lactic acid, but by withholding lime. Virchow also discovered that fresh rickety bones show an alkaline and not an acid reaction, and a careful analysis of the urine also failed to find any lactic acid therein. Finally, the lactic acid, on passing into the blood, must very probably be quickly decomposed into carbonic acid and water, or into lactic salts, and no other acid foods have ever been known to produce disease of the bones. From these and other observations it is plain that lactic acid plays only a very secondary part in the etiology of bone diseases.

3. *The inflammation theory* supposes both these diseases of the bony tissue to be due to a substance floating in the blood, which sets up inflammation therein. The starting point of this theory was a series of experiments by Wegner, who, after pro-

longed administration of very small quantities of phosphorus, observed a thickening of the bones on the diaphyses. From this fact Kassomitz concluded that in rickets also a substance must be present in the blood resembling phosphorus in action, which causes inflammation of the bones with increase of vascularity, during which, and as a result of dilatation of the blood-vessels, the deposit of lime is hindered. And, further, that in consequence of the decomposing action of the carbonic acid of the blood upon the bones, their tissue is broken down. In fact, Kassowitz succeeded, by giving phosphorus in larger quantities, in obtaining bone-modifications precisely resembling the conditions of rickets. Respecting the nature of this inflammatory substance nothing further is known ; but we must assume it to be an infective substance which acts from the blood (haemogenous inflammation of the bones). Virchow has already declared osteomalacia of man to be a parenchymatous inflammation of the bones.

According to Winckel, we have to do with an ostitis chronica with de-calcification of the bones from within, and simultaneous, localised new building up of bony tissue.

Petrone's suggestion is that osteomalacia represents an infectious disease, produced by the micro-organism of nitrification discovered by Winogradsky, and that this bacterium generates nitrous acid in the bones, which dissolves out their lime-salts.

If we sum up all these factors, we have to deal in these bone diseases mainly with conditions of inanition, i.e., of disturbed nourishment of the bony tissue ; the participation herein of infective or inflammatory processes is easily possible ; but the action of lactic acid scarcely comes into the account at all. Upon these lines then we shall proceed to discuss the maladies.

#### A.—OSTEOMALACIA OR BRITTLENESS OF BONE.

(*Softening of the Marrow, Osteopsathyrosis.*)

**Occurrence and Etiology.**—Osteomalacia generally described in veterinary literature as "softening of bone" occurs oftenest in cattle, less frequently in sheep, goats and pigs. It is rarest in horses. It has also been noticed in zoological gardens among giraffes and a few kinds of birds. It is one of the oldest known of diseases among cattle, and was accurately described by Vegetius. It seems to be located in certain districts, where in

some seasons it becomes almost epizoötic in its spread, so as to attract the attention of Government. But it not unfrequently appears sporadically in single farms or stables. Milch-cows are most freely attacked; working oxen or young kine very much more rarely, also old cows. The periods of pregnancy and lactation hasten the development of the disease very remarkably (*osteomalacia puerperalis*). The more milk is yielded, so much the more intense is the ailment; and, again, if the milk-secretion be suppressed, at once an improvement is generally seen. In many districts the cows regularly fall ill 6-8 weeks after giving birth. All this points to an increased out-put of lime on the part of the mother as being a cause of the disease.

But besides this, a deficiency of lime in the soil and fodder are universally blamed. Moor and turf-lands, sandy soil poor in chalk, plutonic rocks and soil deficient in phosphoric acid on the one hand; and sour grasses and pastures on the other, seem to be the most frequent sources of osteomalacia. To these we must add very dry summers, in which there is a lack of fluid to dissolve the lime-salts in the ground and convey them to the grass roots; also feeding on potatoes and turnips, which are vegetables deficient in lime. The chemical investigations of Cantiget are interesting on this point. Tests of various soils gave a lime or chalky average in districts free from this complaint of 245; of only 10-25 in those occasionally affected; and of 9 in those frequently visited by the disease. The corresponding figures for phosphoric acid were 4.1 and 0.9. In correspondence with these figures, we often, if not exclusively, find osteomalacia on those farms where very bulky food is served, such as straw and all kinds of waste of low nutritive quality. Moreover, the small degree of solubility and absorbability possessed by lime-salts, even though, according to analysis, they may be relatively abundant (as, e.g., in fodder of very woody fibre), might also be a predisposing cause of osteomalacia. Finally, the water may assist its development by being insufficiently calcareous.

Some have blamed dirty and ill-kept stalls, but their share in the matter is doubtful. Certainly animals recovering from some other disease (foot-and-mouth) may be more readily infected. On the action of infective matter nothing is at present known, but many etiological factors may possibly co-operate here, as also in rickets.

In man we distinguish a juvenile, a senile and a nervous form of osteomalacia;

**Post-mortem Lesions.**—The pathological changes found in the bones consist mainly in a decalcification and softening from within outwards of the normal bone, with hyperæmia, conversion of the bony substance into a fibrous, soft mass, fatty degeneration and atrophy of the bone cells, enlargement of the medullary cavities, an increasing thinness of the cortex and a change of the medulla itself into a soft and gelatinous mass. The most marked changes are found in the bones of the trunk and of the upper extremities. But the aspect found varies with the severity of affection.

1. *At the beginning* and in slighter cases we find nothing morbid on superficial examination (Roloff), but closer investigation reveals an increased amount of blood in the affected bones and enlargement of the vessels and of the canals which contain them. A section of the bones shows small dots of blood, the marrow is permeated by numerous little extravasations, and the bony tissue round the dilated Haversian canals exhibits slight changes.

2. *In later stages* the hyperæmia increases ; the outer surface of the cortex as well as the bone-section are dotted with blood, the marrow becomes hyperæmic and penetrated by haemorrhages. The medullary cavity itself is enlarged. Upon the diploë and inner surface of the cortex are numerous fine bony plates detached from the mass. The bone also is easier to saw or cut, and its ring has lost its clearness. The microscope shows that the homogeneous structure of the bony substance has vanished ; that in some spots it appears more transparent, the bone cells are enlarged and transparent, have taken on an oval, round or even polyhedral shape, their prolongations disappear and they gradually become transformed into fat cells. The whole represents a transformation in parts of the bony substance into osteoides and finally into medullary tissue.

3. *In its most severe forms* the hyperæmia of bone and marrow continues to increase ; the substance of the cortex becomes spongy, opaque and soft ; cortex and diploë tend more and more to vanish, while the medullary substance grows, presses the cortex from within outwards and penetrates also into the epiphyses. Fractures of the bones then occur. The marrow itself, if general disturbances of nutrition set in at the same time, becomes soft, gelatinous, even watery and has a dirty-yellow colour. The bones diminish in specific gravity, their lime contents being by this time lessened by more than half, but

their watery contents increased. In addition to these processes in the bones, we sometimes find an accumulation of reddish fluid about the joints. Other organs are at first not pathologically changed. But later on we find, along with increasing emaciation and weakness, the signs of cachexia : a hydramic condition of the blood, transudation in the bodily cavities, general poverty of fat, flabby and discoloured muscles, gelatinous infiltration in the cellular tissue, etc.

**Symptoms.**—Osteomalacia does not, as a rule, reveal itself from the beginning in external signs. Indeed, all such signs may be absent for a long time in spite of serious changes in the bones. Thus there may be no pain or emaciation, and the milk-yield may be sustained for an astonishing time. But slight disturbances of digestion often precede the outbreak of the disease, and we notice also a frequent desire to lick various objects ; but this sign may be absent. In cows advanced in pregnancy the disease may appear quite suddenly. Usually it is only the advanced stage which is brought to our notice. We observe then a painful, stiff gait, often on all four feet together, and this lameness sometimes appears suddenly after some exertion or lengthy march. The animals shift from foot to foot, trip to and fro, twitch with their hind legs, evince pain in getting up or lying down, in evacuation or urination, or when pressed upon the shoulders, withers or loins, and at last remain permanently lying. Goats especially seem to be completely lamed and show extraordinary pain on the slightest touch. Under some circumstances these signs are complicated with swellings on the joints with cracking sounds therein, and all the symptoms of articular inflammation, which are supposed, however, not to develop in animals at rest, but only in those which have had exercise, being caused by strain upon the tendons and ligaments, with loosening of the periosteal adhesions and consequent spread of inflammation to the synovial capsule. Later on, fractures, fissures and dislocations occur, and these on the slightest provocation, as in getting up, lying down, turning, slipping and during parturition. Fracture of the pelvis or ribs is most common ; the former occurring especially on the ileum near the hip-joint (acetabulum). Several fractures are frequently found in the pelvis, and in one cow Maris saw no fewer than fifteen. All these are more or less painful and show little tendency to unite. But the much-disputed possibility of the formation of callus seems to have been proved by Roloff's observations.

The ailment generally lasts several months, and during its later course emaciation sets in; the skin grows dry and hard, aprætite falls off, feebleness increases and skin ulceration is brought on by continued decubitus. Hering also observed impiginous eczema of the skin in several cases.

**Osteomalacia of Horses.**—This is much rarer than in cattle, owing to the animals being much better fed and receiving more grain-foods, which are richer in lime and phosphorus. Continuous poor feeding (chaff, bran, bad hay, straw) and deprivation of oats, combined with damp, chilly stables, are the chief causes of the complaint in horses. Probably ricketty foals also sicken later of osteomalacia. The *symptoms* reveal themselves partly in a liability to fracture of bones, and partly in swellings of the two jaws with loosening of the teeth; partly also in stiff, straddling attitudes, much pain when moving, periodical lameness, weakness in the loins, sinking of the back, arching of the breast so as to look quite tub-shaped, and sensitiveness of the muscles to touch. *Post-mortem* shows the same changes as in cattle. In a case described by Belitz the entire vertebral column and the ribs were so soft that they could easily be cut with a knife. The transverse processes of the vertebrae could be rolled up like strips of pasteboard. *Treatment* is the same as for cattle.

**Prognosis.**—The forecast in osteomalacia depends much on whether a change in the animal's outward conditions be possible or not. Very advanced cases may be cured if a change of fodder, a transfer to another district or a rational medicinal treatment (calcium-phosphates, phosphorus) be possible. If circumstances do not permit of such a change, which, for financial reasons, is often the case, then, unless the animals are early slaughtered, they must be regarded as lost, especially if fractures have already occurred. Not rarely an improvement sets in after the period of suckling, but only lasts until the breeding time comes round again.

**Differential Diagnosis.**—The recognition of osteomalacia in its fully developed stages, when fractures have already occurred, is not difficult. But in the earlier stages, when the signs are less characteristic, it may be confused with muscular and articular rheumatism, the more so as complications with these two diseases are not rare. When osteomalacia appears sporadically, a positive differentiation between the two is at first often impossible; but when its spread is epizoötic, a recognition is greatly facilitated by consideration of the etiological factor. According to Anacker, bones affected by osteomalacia give out on percussion a clear and hollow tone, in contrast to the deep

and full-ringing note of sound bones. In goats actinomycotic swellings of the head-bones were formerly taken for osteomalacia; but here the very situation of the disease and the varied nature of the lesions disprove such an idea.

**Therapeutics.**—The treatment of osteomalacia demands, first of all, the removal of its causes. Above all, a change of food must be made and its quality improved. Especially to be recommended are the foods richest in lime, such as leguminous foods, oats, clover-hay, oil-cake, and the straw of beans and peas. A change in the drinking water may also be needful. The best plan is to transfer the animal to another district richer in suitable fodder, but that can only be done in a minority of cases. There is nothing else left then but gradually to improve the soil and its crops by rational manuring and draining. The best manures for this purpose are bone-meal and super-phosphate of lime.

Internally, phosphate of lime must be administered in its most digestible forms. The best in this respect is the prepared (macerate?) bone-meal, which may be mixed with the food in daily doses of  $\frac{1}{2}$  to  $1\frac{1}{2}$  oz. (a tablespoonful with each feed). If this cannot be procured, give calcium phosphate, the same dose. Phosphorus itself may be given in small and long-continued doses (daily  $\frac{1}{2}$  to  $\frac{1}{4}$  grain per cow, in oily solution). Along with these the absorption of lime may be assisted by stimulating appetite. Haime speaks highly of the action of spirits of salt (hydrochloric acid). Roloff suggests only partly milking the cows to prevent their parting with too much lime; but a much better plan would be to withdraw them altogether from breeding while the disease lasts; economical reasons, however, often render both suggestions impracticable.

Since 1888 ovariotomy has been adopted for osteomalacia in women, and partly with good results. This plan has, however, been only very partially tried among cows, and we would suggest that, where the disease makes its appearance epizootically, this method be experimentally tried.

**Osteoporosis.**—In pathological anatomy this name is given to an enlargement of the Haversian canals, accompanied by atrophy and resorption of the compact bony substance, whereby the bones are rendered porous (rarefying inflammation of the bone). We find this phenomenon in the course of osteomalacia. But we do not think there is any justification for regarding osteoporosis as a distinct clinical form of disease. Resorption of the bone by the medulla is also called eccentric atrophy of the bone; whereas with external resorption we have set up a concentric atrophy of

the bone. Both are found with osteomalacia; but they are no more independent diseases than their related form, osteoporosis.

#### B.—RICKETS.

##### *Soft Bones. Dwarfed Growth. Weak Limbs.*

**Occurrence and Etiology.**—Rickets is a disease exclusively of youth, and is most frequently met with in young pigs and dogs, rarer in foals and calves; but it has also been observed in lion cubs and poultry. In some cases the condition is congenital (foetal rickets). The causes correspond generally with those of osteomalacia, and lie chiefly in a lack of lime in the food, a fact abundantly proved by experiment. First comes milk deficient in lime, such as is supplied by mothers suffering from osteomalacia. Among pigs it arises frequently from feeding with large quantities of innutritious and indigestible food which is poor in lime, such as kitchen refuse, or from an exclusive diet of potatoes, as a consequence of which rickets is always most common among young pigs after an abundant potato harvest. In dogs rickets is often caused by feeding them on bread, potatoes and dog-biscuits alone. But an exclusively meat diet may have the same result, if no bones be given also, as was noticed by Röll with young lions. Feeding with bran alone is a most frequent source of rickets in foals (so-called bran-sickness), and may be experimentally so produced. A lack of lime constituents in the soil is also an indirect cause of rickets. Thus Utz noticed that the disease was much rarer upon shell-limestone than upon red-sandstone, granite and gneiss.

The effect of a diet deficient in lime is much increased and accelerated by bad bringing-up. The endeavour by all means to fatten pigs before their skeleton has well formed, their unnatural forcing, the over-crowding of their sties, the foul air, lack of sunshine and absence of all natural exercise, amply explain why rickets is so much commoner in sty-bred pigs than in those which feed at large. Some blame cold as a cause (though probably only of secondary importance) and condemn wet, draughty sties with a cold concrete floor. Gastro-intestinal catarrh may also be a contributory cause, owing to the diminished utilization of the lime-salts in food which it induces, and perhaps in combination with an increased secretion of lactic acid. Again, the continuous and exclusive feeding on butter-milk has been given as a cause among pigs. Finally,

certain breeds of pigs, and especially the finer ones, seem to inherit a pre-disposition to rickets, and since the introduction of the English breeds the disease has plainly been on the increase. Concerning the action of infective matter in rickets nothing certain is at present known.

**Post-mortem Lesions.**—The chief pathological fact in rickets is that the bones remain soft owing to a defective deposit of lime, accompanied by peculiar processes of proliferation in the periosteum and the epiphyseal cartilage.

1. *The periosteum* is hyperæmic and shows upon its inner side a considerable proliferation and thickening of its bone-forming layer, in which process, however, the newly proliferated tissue does not calcify, but mostly remains soft. Not until later does this soft tissue ossify, and then the bone appears thicker and stouter and shows localised hyperostoses. Such periosteal proliferations occur especially on the points of attachment of the muscles, for instance, in pigs, at the insertion of the psoas magnus and iliacus internus into the femur, and also often on the point of the os calcis. Moreover, the thickened periosteum may become detached from some bones by the pull of the muscles, from the scapula, for instance, in pigs.

2. The chief changes in rickety bones show themselves on the epiphyseal border, and consist in an immense proliferation of the *epiphyseal cartilage* without proper calcification. Whereas normally the cartilage between epiphysis and diaphysis is separated from the latter by two thin parallel layers, the proliferation-layer and the ossification-layer, in rickety bones the former of these is excessively developed, while the latter is backward in its growth. The two no longer run parallel, but are serrated and overlap. We find also in the proliferated cartilage-layer scattered foci of formation of medullary space and calcification: the formation of medullary space in rickety bones is consequently advanced beyond the line of ossification. This abnormal proliferation of the epiphyseal cartilage leads to the thickening and swelling of the epiphysis, which only ossifies during prolonged course of the disease, and, besides that, also to curvature and bending of the longer bones, and finally to displacement of the epiphysis, because the connection between epiphysis and diaphysis is loosened.

From this proliferation of the cartilage and periosteum there arise numerous deformities in the bones. They are retarded in their growth (dwarfed growth), are short, broad

and thickened, and show protuberances, especially near the joints (so-called double joints) and are easily bent. Peculiar "badger-legged" or bow-legged curvatures occur on the extremities, lumpy elevations or large depressions upon the vertebral column (the upward deflections, causing "fish-back," are called cyphoses, and the downward ones, producing a sunken back, are lordoses, while the lateral curvatures are named scolioses). Upon the thorax a so-called pigeon-breast may develop, while the pelvis may become contracted in diameter. The bones themselves suffer infraction and fracture. Very frequently the union of the ribs with the costal cartilage is implicated; the abnormal proliferation of cartilage producing here the so-called rickety necklace (or rosary). Schütz also observed peculiar rickety changes on the skulls of dogs; whereas sound pups were born with their skull-bones completely closed, he found interstices, or fontanelles, between the several bones, these latter also being mostly attenuated. Finally, the joints likewise participate secondarily in these changes in the bones; for, consequent upon the softness and flexibility of the latter, the joints are more exposed to wrenchings and twistings than usual, so that rickets is often complicated with articular inflammation.

**Symptoms.**—The development of rickets is always gradual and slow, lasting usually several months. Its phenomena in the various domestic animals are mainly as follows:

1. In swine a desire to lick things is said often to be a preliminary sign of rickets. But usually a stiff, constrained gait is the first to be observed (so-called "stiffness of sucking pigs"); the animals also arch their backs, lift the affected leg and can hardly be got to stand up. Bony tumefactions show themselves on the epiphyses near the joints, as well as on the ends of the ribs near the sternum, which are painful when touched. Further on appear curvatures in the bones; the feet are twisted forwards or backwards, the legs becoming bandy or bowed, and the astragalus often touches the ground. A sharp pressure upon the curved bones sometimes produces a crackling noise. The vertebral column is either bent forward (fish-back, cyphoses), or sinks downwards (hollow back, lordoses), or is bent sideways (scolioses); the pelvis is laterally contracted upon the hip-joints, and may later become a cause of trouble in parturition (rickety pelvis). Occasionally we notice a

simultaneous swelling of the nasal and jaw bones, as well as of the lower jaw, thus impeding both mastication and respiration ; this condition represents what is called snuffling sickness. The animal's growth is consequently retarded and often remains dwarfed ; the change of teeth is also gradually delayed. These curvatures of the bones are soon complicated with articular inflammation, with swellings and protuberances on the joints, especially on the ankle, carpal joint and pastern. As the disease advances appetite falls off, the animal grows very thin, has diarrhoea, becomes quite lame in the hind limbs and at last remains constantly lying. At times impetiginous skin-eruptions and bronchial catarrh may be perceived, and the animal finally perishes of cachexia.

2. In **dogs** rickets is manifested by bony tumefaction and especially in button-like thickenings on the border-line between the ribs and the costal cartilages (rickety necklace), curvature of the extremities (bandy legs, bow legs), chiefly of the radius and ulna forwards ; in a helpless, painful gait, treading with the whole foot upon the ground, so that even the os calcis touches the floor ; also in swellings upon the ends of the joints (double joints), development of a "pigeon-breast" (increased diameter from vertebral column to sternum and lessening of the transverse diameter of the thoracic cavity), etc. The change of teeth is often disturbed and retarded, the teeth are abnormally small and show defects of enamel. The dogs are also subject to eruptions.

3. In **horses** a stunted growth and fatigue] on the slightest effort are first noticeable. The bulging of the bones occurs also, especially upon the epiphyses of the limb bones, particularly the shin-bone and bones of the head. Upon the latter they seem to be caused by the pressure of the halter and mostly upon the nasal bone. Swelling of the upper jaw is also not rare, produced probably by pressure of the molar teeth. This may even cause narrowing of the nasal orifice, difficulty of breathing and chronic nasal catarrh. The curvature of the leg-bones is usually not so extreme as in pigs and dogs ; and yet one may sometimes see foals whose forelegs are bent like those of a dachshund. But rickety foals often display a "goat-footed" appearance, giving way at the fetlocks and placing their hind feet on the ground in a way resembling bears. The joints also are not unfrequently thickened, the hocks (a bulging

of the bone like spavin) and the knees especially; and the bones show great liability to fracture. The back is often either arched, or sunk, and the process of dentition is delayed. Now and then the horse manifests pain by shuffling about on its feet, twitching the hind-legs or groaning. Finally, rickets appears to cause a predisposition to eczema, bronchial and intestinal catarrh, decubitus and swelling of the thyroid gland.

4. In cattle the phenomena of rickets consist, as in other animals, in thickening of the bones, especially of the carpal and tarsal, curvatures of the back and leg-bones (X-legs, knock-knees), rickety necklace, rickety pelvis, painful gait and persistent lying down.

5. Among poultry, according to Zürn, rickets occurs most frequently in fowls three to six months old, but is rarer in pigeons, geese and ducks. Its symptoms are continual sitting, difficulty in walking, knotty swellings on the joint-ends of the extremities and wing-bones, softness, flexibility and even curvature of the bones, arching of the breast-bone, etc.; also in wasting away and in poverty of blood.

**Prognosis.**—The forecast in rickets, as also in osteomalacia, is favourable when it is possible to remove its causes. By rational treatment very advanced changes in the bones of animals still young may be repaired. But abnormal deformities, sunken backs, rickety pelvis or thoracic malformation are naturally past all cure. With less valuable animals, therefore, such as pigs, it is better in such cases to slaughter them promptly, as such deformed creatures are later of little economic use.

**Differential Diagnosis.**—Rickets can only be mistaken for pyæmic arthritis and acute articular rheumatism. But apart from the bulging and bending of the bones, these two diseases are distinguished by their sudden occurrence, and rapid, febrile course; and pyæmic articular inflammation by its suppurative character.

**Therapeutics.**—The chief remedy for rickets is a prompt and persevering administration of phosphorus. We prescribe for older foals single doses of  $\frac{1}{2}$  grain; for dogs  $\frac{1}{100}$  grain of phosphorus dissolved in oil or in cod-liver oil. For horses

we would take, say, phosphorus 4-8 grains; cod-liver oil 10 oz., daily one tablespoonful in bran-mash. For dogs and pigs: phosphorus,  $\frac{1}{2}$  grain; cod-liver oil, 10 oz., daily a tablespoonful (for small animals a teaspoonful). To poultry give the last named solution in drops. In the second place comes as a remedy an increased supply of lime. Prepared bone-meal mixed with the food, either a tea or table-spoonful, according to the animal's size; and for dogs also calcium phosphate in daily doses of 8 to 75 grains. These have both proved themselves efficient. At the same time change the food if possible, but avoid over-feeding, so as to remove any gastro-intestinal catarrh, and improve the conditions of breeding. With pigs a more natural manner of life must be above all things permitted in place of their unhealthy sty-existence, and they must be let out into the air and sunlight as much as possible. According to Merz, the dwellers near Lake Ammer cure their rickety chickens by feeding them on fish. In this case, not only is the food nitrogenous, but the fish-bones also contain much lime.

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#### BRAN OR KRÜSCH DISEASE.

The bone-disease of horses described under this name is among foals of essentially rickety origin. It is caused by an exclusive diet of bran, and, therefore most commonly appears among millers' horses. The earliest *symptoms* are digestive troubles, constipation, weariness, profuse sweating during work, etc. These are complicated with swellings of the bones, especially near the carpal and tarsal joints, with lameness and passing attacks of pain, which favour the head-bones and particularly those of the nose and jaws; also with difficulty in taking food and in swallowing, with loosening of the teeth, etc. Finally, the animals perish of cachexia and anaemia. When the disease attacks the young, it is not to be distinguished from rickets. Bran is a food quite as unsuitable for foals as potatoes for young pigs, and an exclusive bran diet can have none but injurious consequences, apart from the fact that it contains much phosphoric acid. Indeed Pütz considered Krüsche sickness as a kind of chronic phosphorus-poisoning, corresponding to the phosphor-necrosis of the upper and lower jaws in man, and traced it to the great proportion of phosphoric acid in bran. But this hypothesis is quite untenable. It is known that only free phosphorus, and not its acid, exercises an inflammatory action upon the bony tissues. In older animals this disease is probably identical with osteomalacia. Some supposed cases may really have been actinomycosis.

**Snuffing Sickness.**—Under this name several authors have described a disease of pigs, which is evidently not of uniform character.

Sometimes the primary ailment seems to be rickets, at others a chronic haemorrhagic, suppurative nasal catarrh, possibly of infectious nature, and again tuberculosis, actinomycosis or sarcomatosis. This labelling of varied complaints with one name renders impossible any definite classification of snuffling sickness, and it would be better to drop the name. The aspects common to all are an audible snuffling respiration, which, at first intermittent, becomes later continuous, a swelling of the upper jaw, thickening of the nose, as well as shortening and distortion of the snout.

A. In the *catarrhal form* we also perceive at first a sero-mucous, and later a bloody-purulent nasal discharge, with frequent bleeding from the nose, which latter temporarily relieves the breathing. We find also conjunctivitis, cough, difficulty in taking food, retching and, when the course is prolonged, also emaciation and death from cachexia or suffocation. *Post-mortem* reveals purulent, haemorrhagic inflammation of the nasal mucous membranes, with extraordinary distortion and extreme atrophy of the turbinatae and ethmoid bones. Schneider seeks the origin of this catarrhal form of snuffling sickness in a congenital, rudimentary structure of the bones just named, which promotes accumulation of foreign matter in the nasal cavity and a consequent inflammation of its mucous membrane.

B. The *ricketty form*, which alone really belongs to this chapter, is also marked by rickety affection of the extremities. In the cases observed by Haubold he always found inflammation of the joints, and Lafosse noted also all the symptoms of rickets. Moreover, in foals, quite similar swellings of the nose-bones and their surroundings occur during the course of rickets, accompanied by difficulty of breathing. Respecting the other forms of snuffling sickness nothing positive is yet known, and further investigation is to be desired.

The *treatment* of the disease varies with the primary affection, but is in general not very successful, and early slaughter is therefore advisable.

**Acute Infectious Osteomyelitis.**—By this name a febrile, infectious disease of young people is known, which occurs in human beings through South Germany, Switzerland and on the north German coast, and in which the long bones (femur, tibia and, more rarely, the radius and ulna) show purulent and ichorous foci of inflammation. It is specially distinguished as a genuine osteomyelitis from the secondary osteomyelitis which occurs in the course of other infectious diseases (typhus, scarlet fever, measles, pyæmia, tuberculosis, cystitis, etc.) and in poisoning. The genuine infective osteomyelitis of man is produced by certain bacteria, viz., *staphylococcus pyogenes aureus* and *albus*. Respecting the presence of osteomyelitis in our domestic animals as a clinical form of disease nothing precise is known, and we still lack exact and authoritative research. Sticker has furnished a synopsis of the pathological changes in the medulla of the bones during the several diseases of horses (1887).

## CHAPTER XII.

### TRICHINOSIS IN SWINE.

**Natural History Notes.**—The trichina (*trichina spiralis*) is a small thread-shaped round worm of the family of hair-worms (*trichinidae*), which was imported into Europe during the thirties of last century, probably by Chinese pigs, and quickly spread all over the world. Paget discovered, and Owen first described and named the muscle-trichina in 1835. Leidy found it in pigs in 1847, Leuckart and Virchow proved the presence of intestinal trichina, and Zenker demonstrated the trichinosis of man in 1860, in Dresden. Two development forms of the trichina must be distinguished: the sexually ripe intestinal trichina and the sexless muscle-trichina.

1. *The intestinal trichina* lives on the superficial layer of the mucous membrane of the small intestine, is straight and has a pointed head. The male is  $\frac{1}{10}$  inch long and the female  $\frac{1}{2}$  to  $\frac{1}{4}$  inch. Its digestive canal consists of mouth, oesophagus, stomach, intestine, anus and a cleft-like cloaca; the generative organs of testicular-tube, spermatic duct and male sexual opening on the one hand, and upon the other of the female ovary, uterus, vagina and vulva. A female produces on an average about 1,500 living young during her life of from five to six weeks.

2. *The muscle-trichina* represents the larval form of the above, from whose embryos it develops. Its length is up to  $\frac{1}{4}$  inch; it possesses a pointed head and a round, cloven hinder part (cloaca). It lies in the muscles in oval to round capsules, from one to four in each, rolled up in serpentine, spiral or annular form, or in the shape of a pair of hand-cuffs. The life-duration of a trichina may, in a pig, amount to 11 years and in a man to 27 years.

**The Development of Trichines**—according to the in-  
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vestigations of Leuckart, Zenker, Pagenstecher, Haubner, Virchow and others—is as follows. The starting point is the consumption of muscle-flesh containing trichinæ. It is still uncertain whether embryos of intestinal trichinæ evacuated with the faeces are directly ingested. Attempts to inoculate by feeding with intestinal trichinæ have only very occasionally succeeded. On the other hand, it is not impossible that undigested morsels of flesh containing them may be passed with the dung, which, on being consumed by other animals, cause trichinosis; but in this case again the muscle-trichina, and not the dung, is the infecting medium. We can distinguish four stages of development: 1. *The stage of embryo-formation in the intestine*, beginning on the seventh day after their reception. The encapsulated muscle-trichinæ are in 24 hours set free by dissolution of their surrounding capsule; in another 24 to 48 hours they are sexually ripe, copulate, and bring forth living embryos on the seventh day, reckoning from that on which the meat was eaten. According to later investigations of Cerfontaine, confirmed by Askanazy, the mature trichinæ do not all remain in the intestines, but a part of the impregnated females quit them and enter the lymph-vessels and lymph-follicles of the intestinal wall, and even pierce to the mesenteric lymph-glands, whence the larvæ to which they there give birth are carried through the lymph-passages into the blood itself, and thus into the muscles. 2. *The stage of immigration of the embryos into the muscles*, which lasts from two to three weeks. From the seventh day forward the embryos begin to perforate the walls of the intestine in which they were born and to work their way into the muscles, partly through the vascular system and partly along the connective tissues, the duration of such emigration being nine to ten days. 3. *The stage of encapsulation in the muscles*, lasting from four weeks to three months. On reaching the muscles the embryos lie quiet for about 16 days and then change into muscle-trichinæ. The muscular fibres lose their diagonal striation, the sarclemma tube becomes dilated like a spindle around the embryos which have penetrated into the middle of the fibres; the muscle-nuclei multiply, while the contents disappear, the connective tissue is infiltrated with small cells, and the capillaries are much distended. Finally, regular layers of cells form round the trichinæ, which thicken into a round, lemon or eye-shaped capsule of connective tissue, whose two pointed ends are darkened at the poles. At this point the flesh begins to be dangerous owing to its infective

quality. 4. The calcification stage of the capsules, lasting from three to eighteen months. After the third month the enveloping capsules begin to calcify, so that the trichinæ themselves can no longer be seen. The calcified capsule can then be perceived by the naked eye on a thin section as a small light spot, about  $\frac{1}{8}$  inch long. Sooner or later the trichinæ also calcify or undergo fatty degeneration. Trichinæ have been found in the adipose tissue (bacon-sides) as well as in the muscles, both in their free and their encapsulated condition.

**Occurrence.**—Up to the present trichinæ have been observed in man, in swine, rats, mice, dogs and cats; also in the wild boar, fox, bear, marten, pole-cat, domestic fowl, turkey and jay. They have, moreover, been brought to development by feeding in very many of the mammalia, such as the horse, ox, sheep, dog, rabbit, ferret, hare, raccoon, badger and guinea-pig. Their favourite muscles are the crura and the muscular part of the diaphragm, the tongue, muscles of the larynx, masseters, those of the eye, the temporal and intercostal muscles, also those of the neck and trunk and the twin or gemelli muscles. So far no muscle-trichina have been discovered in the heart or smooth muscles, but they have been found in the adipose tissue near muscles. The number of individual trichinæ in the different muscles varies greatly; as many as 1,500 may be present in 15 grains of flesh.

The statistics of the spread of trichinosis among pigs vary greatly in different countries, but the disease is generally more common in north than in south Germany. In Prussia about one pig in every 2,000 seems to have trichinosis according to the figures given, but in Germany generally the proportion is only one in 10,000. In America, on the contrary, the percentage is much higher, the investigations of Billings (in Boston) showing one in twenty-five, or 4 per cent. As regards the ham and other swine-flesh imported into Germany from America, we may assume as the result of present researches that an average of 2 to 3 per cent. contains trichinæ (a maximum of 8 per cent.).

**Pathogenesis.**—The manner in which swine are infected with trichinæ has, up to the present, not been satisfactorily explained. As the main source of infection trichinous rats and mice, which the pigs greedily devour, are chiefly suspected, and such trichinous rats are mostly met with in horse-slaughtering

establishments and abattoirs. Thus, among 18 of the former, Leisering found 14 so infested; and Franck found 9 per cent. of rats brought from flaying-houses to be trichinous, and Huller 8 per cent. of those gathered from various places. There must, therefore, be some connection between rats and swine. But pigs may also be directly infected by feeding them with the flesh of other pigs which have died of the disease, or with the offal and slaughter-house refuse of other infected animals; possibly also indirectly by taking up the dung of swine containing undigested morsels of trichinous flesh. According to Hertwig's observations it seems that successive immigrations of trichinæ may occur, as they are found in various stages of development in the muscles. We must await further researches on the possibility of infection by the embryos of intestinal trichinæ passed out with the faeces. The occurrence of congenital trichinosis is more than doubtful.

**Symptoms.**—Apart from the cases of experimental feeding, no symptoms of trichinosis have yet been observed in swine during life, probably because such symptoms, being so little characteristic, have been overlooked by owners during the period of immigration, or confused with other diseases. And later, when the immigration is ended, the general condition of the trichinous animal is in no way disturbed. Usually when the pigs are killed the time of immigration is long since over, which explains the fact that, in spite of the proved presence of great quantities of trichinæ in the muscles, no signs of illness could be perceived immediately before slaughtering. Moreover, the absorption of a small number of trichinæ causes no morbid symptoms. The aspect of trichinosis, as produced experimentally in pigs by feeding them on trichinous flesh, allows us to distinguish two stages, that of an intestinal affection, as well as the subsequent derangements in the muscles.

1. The *affection of the intestinal canal* occurs at the end of the first week and beginning of the second after infection. It betrays itself by loss of appetite and spirit, in arching the back, tension of the abdomen, continued diarrhoea with signs of colic and much debility; in a few cases vomiting was also noticed. In young animals, or if the quantity of trichinæ absorbed be very large, the disease may prove fatal even in this stage. On the other hand, if such quantity be only moderate, the animal may recover.

2. The *symptoms* are observed in connection with the muscles

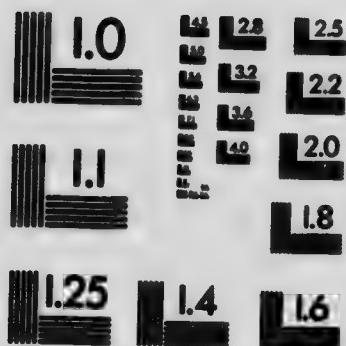
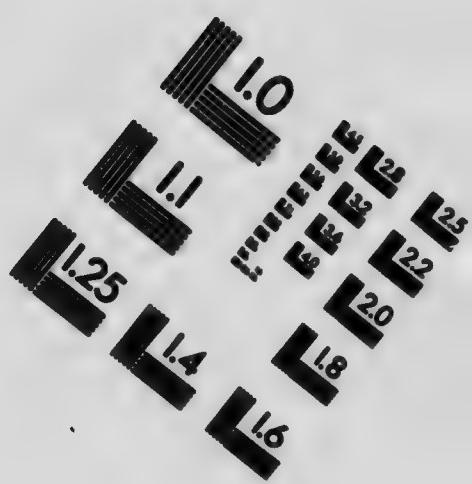
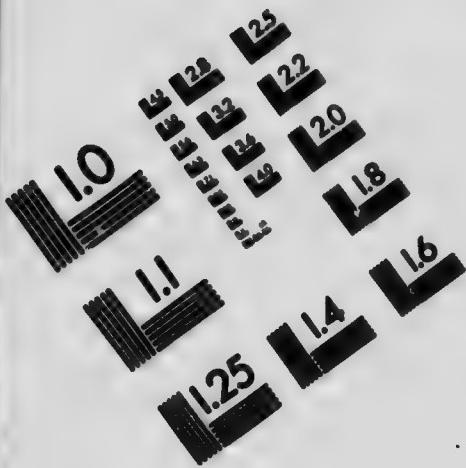
with the immigration of the embryos into their fibrils, and the development of inflammatory conditions; they occur therefore in the second and third weeks. The animals display a peculiar itching, which makes them rub and scratch themselves; also a stiff and constrained attitude of the limbs, which may amount to lameness and weakness; they manifest pain and rapidly lose flesh and constantly lie down. Difficult mastication and swallowing are also observed, difficult and painful breathing, a feeble, hoarse voice, and also oedematous swellings in various parts of the body. Usually a complete recovery is made after the condition has lasted four to six weeks, when the animals quickly grow fat again. Only in rare cases is the result fatal.

**Trichinosis in Dogs.**—A young dog, one year old, which was fed on very trichinous pork, manifested signs of restlessness and bloody diarrhoea after two weeks, and a little later symptoms of lameness and spasms. The neck-muscles more especially were attacked by tonic-clonic spasms, which sometimes spread over the whole body. The hind legs were completely paralysed, and the temperature rose to 103° Fahr. On dissection, numerous migratory trichines were found in the muscles. Another very old dog remained quite healthy in spite of the same food (Diugay).

**Diagnosis.**—As a rule, it is impossible to recognise trichinosis during life, its symptoms being so little characteristic and so much like those of intestinal catarrh or muscular rheumatism, and because the trichinae, once encapsulated, cause no further morbid disturbances. But the demonstration of the disease is very easy after slaughter. Even with the naked eye the calcified capsules can be detected in thin slices of the flesh as fine, light-coloured spots. But a positive diagnosis is only possible by the aid of a microscope, for which an enlargement of 40 times is most suitable. A glass of only ten diameters is enough for the experienced observer, but is not to be recommended for others. The simple method employed is to clip thin sections with sharp, curved scissors out of the muscles most frequently attacked, and if possible close to their place of insertion, parallel to the line of their fibres. Place these on the glass plate which serves as object bearer, spread them out and cover with a similar glass, squeezing them so as to be transparent. According to the size of these glass plates, a number of sections may be examined at once. To ensure a positive result, it is usual to cut at least six specimens from different muscles (midriff, tongue, larynx, eye, intercostal and neck) and to make six preparations of each. Ac-

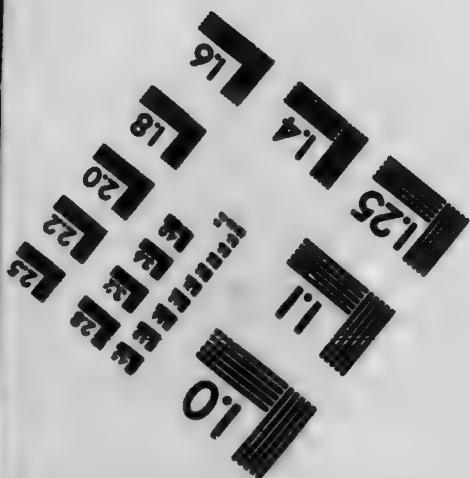


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cording to the latest enactment in Prussia, half these sections must be cut from the crura of the diaphragm, which is usually richest in trichinæ. The occurrence of the worm in the adipose tissue (bacon-side) also makes an examination of this part necessary.

**Differential Diagnosis.**—Microscopical examination of the muscles for trichinæ not seldom leads to confusion with other and similar formations in the muscles. Among these are first the so-called concretions. These are greyish-white bodies, about the size of a millet-seed, which often permeate the muscles in great numbers and apparently vary very much in their nature. Sometimes they consist of crystals of lime, tyrosin, margarin and stearin, at others they seem to be calcified cysticercæ or new growths. At one time they were supposed to be concrements of guanin. But they all differ from trichinæ in being considerably larger, in their different shape and in the absence of a round-worm in their interior. In particular they lack the eye-shape of the trichinæ-capsule, its darker poles and sharp contours. Trichinæ may also be mistaken for Miescher's sheaths (or tubes); but these possess no capsule, and are longer in shape with blunt ends, are tightened up into sections (like sausages), are mostly also larger and consist of dark, granular matter, which, under a more powerful glass, can be seen to be crescent-shaped bodies; finally, the diagonal striation of the muscular fibres immediately surrounding these Miescher's sheaths remains quite unimpaired. Furthermore, trichinæ have been confounded with the spores of actinomycetes (Dunker), which occur in the form of round or bean-shaped objects, with radiating stripes and thin centre with thicker edges, and reveal, on being crushed, a club-shaped mycelium which strongly refracts the light. The actinomycotic nature of these objects has, however, been contested by Johne. There are also certain helminths inhabiting the muscles which may be mistaken for trichinæ, especially the distomata which lodge in the laryngeal muscles and in the crura of the diaphragm. But these may be recognised by their two suction-pores, their cylindrical form and lively movements. Finally, we would mention gargols, echinococci, rhabditides and the embryos of round worms, haplococcus reticulatus, etc.

**Prophylaxis.**—As destruction of the trichinæ in the living animal is altogether impossible, there can be no question

of treatment for the disease ; but, on the other hand, it may be prevented by proper precautions. Among these is the complete destruction of the carcases (by high temperature) of all trichinous animals, for which end efficient meat-inspection is of the highest importance. To this we would add the extirpation of rats and mice in the pig-sties, the prohibition of all pig-keeping in slaughter-houses and knackers' yards, and finally a discontinuance of flesh as food for pigs.

**Trichinosis in Man.**—Since trichinosis was first established as a disease of mankind in 1860, there have been more than 50 epidemic outbreaks. The *symptoms* of the disease in man are very variable. During the first eight days they are usually confined to those of gastro-intestinal catarrh (loss of appetite, nausea, vomiting, diarrhoea, fever, colic, and occasionally attacks like cholera). But from the tenth day muscular symptoms become most prominent (pain in moving the muscles, swelling and tetanic hardness of those affected, lying down with bent limbs, difficulty in chewing and swallowing, hoarseness, aphonia, hard breathing, oedema of the eyelids, of the face and extremities, and fever). *Treatment* (purgatives, benzine, santonin, oil of turpentine, glycerine) has hitherto always proved of no avail. The *infection* always occurs through eating raw or half-raw swine's flesh. *Prophylaxis* consists therefore in avoiding such food : Pork, when thoroughly boiled through or roasted through is perfectly harmless, because the trichinae perish at a temperature of  $143^{\circ}$ - $167^{\circ}$  Fahr. in consequence of the coagulation of their albumen. But the boiling must be long continued. A piece of beef, 6 to 7 lbs. in weight, according to Vallin's experiments, only attains at its centre a temperature of  $194^{\circ}$ - $2.2^{\circ}$  Fahr. after boiling for four hours, whereas, after one hour it has only reached  $122^{\circ}$ . For ham, three hours' boiling are not enough. To roast beef a heat of only  $122^{\circ}$ - $140^{\circ}$  is needed, and for roast mutton  $118^{\circ}$ - $133^{\circ}$ ; and these roasts are therefore no guarantee against trichinosis. But the trichinae may also be destroyed by thoroughly *pickling* the meat for several weeks, or by *hot smoking* for at least eight days. Finally, a brief exposure of the meat to a pressure of twelve atmospheres is said to kill the trichinae. But that insufficient pickling, even when continued for several weeks, does not kill them is proved by the feeding experiments made with imported American hams (Janssen, Rievel and others). Living trichinae were also found in American hams (Höfnagel and others). The numerous negative feeding experiments made with trichinous American hams (Fränkel, Klaphacke, Schenk, Wagner, Hintzen, Schmitt, Browner and others), show, on the other hand, that the vitality of the trichinae is very much enfeebled by pickling. That trichinous meat is rendered innocuous by long exposure to cold, as maintained by Bonley and Gibier, is, according to later tests, very doubtful. Gibier has recently shown that after two hours under a cold of  $13^{\circ}$  Fahr. below zero, the trichinae had not suffered. As against these more direct methods of protection, microscopical examination of the meat is of secondary importance as a safeguard against infection. But it may be recommended in districts where the above rules are neglected.

## CHAPTER XIII.

### THE MEASLES OF DOMESTIC ANIMALS.

#### I.—MEASLES IN PIGS.

**Natural History Notes.**—This disease, which was formerly known as, miliary fever or pearl-rash, is produced by the bladder-tail parasite of the connective tissue, or gargol (*cysticercus cellulosæ*), which is the larval form of the *tænia solium* of man. The connection between the hermit tape-worm of man and the *cysticercus cellulosæ* was first established by Küchenmeister and Haubner by means of feeding experiments. Measles, therefore, in pigs depends in its frequency upon the occurrence of *tænia solium* in man. The swine are infected by absorption of human excrement containing proglottides, and owing to their preference for such excrement, pigs are of all domestic animals most liable to this form of infection. Those pigs which feed in the open and on fields largely manured with refuse from city middens and cesspools, or which live in dirty sties situated near human privies, most frequently suffer. The disease consequently varies much in frequency according to the manner in which swine are kept.

The development of *cysticercus cellulosæ* takes place when the eggs of *tænia solium* pass into the stomach along with the proglottides—which latter may already be in a putrid condition—and are there digested by the gastric juices. The embryo (which has six hooks) is thus set free, bores through the intestinal wall and wanders on, partly along the connective tissue, and partly as it is carried by the blood. These immigrations, however, only occur with younger pigs, up to six months of age; older ones not being infected. When once the embryo has become affixed, it loses its hooks, becomes bright in the centre, proliferates outwardly and changes into a vesicle, upon whose inner wall a peg-shaped body grows, which is the rudi-

ment of the scolex. In 20 days the parasite is the size of a pin-head, the future head being indicated by a dull dot; in 40 days it has grown as big as a mustard-seed, the head now being distinctly seen, though the suction-cups and hooks are still in the rudimentary stage. At the end of 60 days it has attained the size of a pea, hooks and suction-cups are clearly visible, but there is no neck; and after three months the cysticercus is fully developed and able to infect mankind. Round its location a reactive inflammatory process is set up, which leads to the construction of a capsule of connective tissue. It must not be overlooked that among those forming the same immigration much variety of development occurs, some cysticercæ dying early and becoming calcified, while the rest go on to perfect growth. According to Ostertag's observations, those which find their way to the liver and lungs usually die off soon.

**Occurrence.**—The favourite localities for cysticercus cellulose are the interstitial connective tissues of the muscles, of which those of the tongue, heart, neck, pelvis, flanks and upper thighs are most frequently chosen; also the subcutaneous connective tissue, the brain, spinal marrow, eye, liver, spleen, lungs, body cavities, etc. It may also be found in the fat. Not unfrequently all the body organs are inter-penetrated by thousands of these parasites, so that there may be from three to four for every scruple of flesh. It is a much rarer thing to find them isolated. The fully developed cysticercus varies in size from a pea to a bean and presents the appearance of a bladder of dull white colour and semi-solid consistence, which has both a head and neck. The former is an opaque dot, usually curved inwards, giving the bladder somewhat the form of a kidney, whereas, when the head is convex, it has more the form of a round-bellied bottle. Upon the head are four suction discs, a rostellum and a double circlet of hooks with 26 to 32 hooks. The bladder is usually enclosed in a capsule of connective tissue, which is only absent when in the serous body cavities. These vesicles swell out when boiled and creak on being cut; those that have died undergo fatty degeneration and calcification. The muscles round about remain at first normal, but later, as the cysticercæ grow numerous, become greasy and watery.

The frequency of measles varies much in different districts. Official figures give an average for the kingdom of Prussia of 3 infected swine to 1,000 healthy ones. In other countries the

average is higher ; thus, in Odessa, 371 out of 3,060 pigs examined were found to be affected, or about 12 per cent.

**Symptoms.**—These are by no means characteristic. Very many pigs, although highly infected, reveal no morbid symptoms whatever during life. But in general the following are cited as signs of the disease : a hoarse, rough, croaking grunt, falling out of the bristles, faintness and depression, lessened appetite, paleness of the oral mucous membrane, anaemia and emaciation, oedematous swelling of the head, neck, etc., diarrhoea, weakness, paralysis and finally death from exhaustion. In a few cases death occurs very quickly, when, for example, an unusual quantity of the tape-worm eggs has been absorbed. In contrast to these very undecided general symptoms, the aspect of the disease is much more clearly marked when localised upon any special organ of importance. Thus we not seldom observe, after immigration of the parasite into the brain, severe cerebral phenomena in the form of epileptiform spasms, paralysis of one or both sides, mania and raving. Or if it attain the eye (beneath the retina, in the vitreous humour, the lens or the chamber of the eye), we notice blindness. When the parasites are too numerous in the tongue, that member is paralysed, and the animal cannot take its food ; and excessive localisation upon the serous membranes causes peritonitis and pleuritis. Finally, sudden and apoplectic death has been known to occur.

**Diagnosis.**—The identification of measles in swine is only possible during life when the cysticercæ can be felt or seen on the under side of the tongue near its frenum, or upon the inner surface of the eyelids, where they appear through the mucous membranes as hard nodules. It is recommended, when the former is not the case, to make an incision lengthwise in the tongue in order to lay bare any that may be present. It is possible that in future the ophthalmoscope may be of service in this diagnosis. But one cannot conclude from the absence of the cysticercæ in the tongue that the animal is entirely free ; for out of 41 diseased animals Railliet only found the parasite there in 31 cases.

But the cysticercæ cellulose are easily recognised in the slaughtered carcase, difficulty only arising when they are already dead and degenerated. But diagnosis is in all cases assured by microscopical identification of the head, hooks or external

case. But Ostertag's investigations have established, with regard to the diagnosis of dead cysticercæ, that the discovery of so-called lime-corpuscles in a calcified or caseous muscle-focus is a positive proof that such focus has been produced by a bladder-worm. To certify the presence of cysticercæ in minced or sausage-meat Schmidt-Mülheim recommends, seeing that the ordinary microscopical method is tedious and detailed, that such flesh be digested for several hours in six to eight times its weight of artificial gastric juice at 104° Fahr., whereby all but the head and its circlets of prehensile hooks is digested, and these, gathering at the bottom of the glass, can then easily be detected by a powerful lens. This artificial gastric juice is prepared by pouring glycerine over the chopped mucous membrane of a pig's stomach and allowing it to act for some time, when a small quantity of this pepsine-glycerine is replaced by a  $\frac{1}{4}$  per cent. solution of hydrochloric acid.

**Prophylaxis.**—The precautions against measles in pigs to be adopted consist in preventing them from consuming human faeces, cleansing and improving their sties, avoidance of pasture-feeding, of human privies and dung-heaps. This of course, refers only to the young pigs up to six months. To this should be added, if possible, the treatment and cure of all human beings suffering from tape-worms and extirpation of the latter. No treatment of the sick pigs themselves is of any avail.

In most states this is a disease of pigs for which a warranty can be demanded. The time-limit of such warranty in Bavaria, Prussia, Hesse, Austria and Waldeck is eight days, in Württemberg, Baden and Hohenzollern 28 days, and in Saxony 30 days.

Man is only infected by measly pork when it is eaten raw or half raw, and in the latter connection half-cooked sausages are particularly dangerous. The prophylaxis is in such cases very simple, as with trichinosis, and consists in thoroughly boiling or roasting the meat quite through. According to Perroncito's tests, the cysticercæ perish if exposed longer than a minute to a heat of 122° Fahr. They are also killed by being well smoked, pickled or dried. As a result of their loss of water they shrivel up to the size of a pin-head, and become hard bodies which crackle between the teeth. The legal enactments concerning the consumption of such flesh vary in different countries; but its use ought to be strictly prohibited, or restricted to industrial processes, such as soap or glue-manufacture. This prohibition should, in our opinion, extend also to the fat and bacon, which are not rarely infected, and which people often consume raw. On the other

hand, meat only slightly measly (*i.e.*, containing only solitary cysticercæ) might be admitted to consumption under certain restrictions. These should consist in boiling under proper supervision, and only allowing it to be sold after duly informing the buyer of its nature and how it should be treated. The dangers arising in man from eating measly pork are first, the incorporation of one or more tape-worms, which may cause disturbances of digestion and nutrition and lead also to psychical affections; also the self-infection, through the eggs of *tænia solium*, with a formation of cysticercæ in the brain, eyes, etc., producing functional disturbances of the brain, blindness or even sudden death. In consequence of the care exercised in inspecting meat in our larger cities, *tænia solium* has become very rare in man. In Berlin, for instance, the common tape-worm is *tænia inermis*.

## II.—MEASLES IN CATTLE

**Natural History Notes.**—The measles of cattle represent the sexless larval stage of *tænia medio-canellata* (*saginata*, *inermis*) of man. It is rarer than swine-measles, but much commoner than is generally supposed. Its development also depends on the consumption of the proglottides and eggs discharged with human excrement. In by far the larger number of cases the parasite infests the inner and outer masticatory-muscles, and in a secondary degree the heart, the proportions being about 9 to 1. More rarely the tongue, the muscles of the neck and breast, and the adipose tissue are attacked; and most rarely of all the liver, lungs, brain, kidneys etc. A further distinction between the disease in cattle and in pigs is that the parasites are not so numerous as the cysticercus *cellulosæ*, but rather occur singly. Sometimes only a solitary one is found in the muscles of the jaw. And yet in one case Hering counted 300 in half-a-pound of ox-flesh. Of 389 measly cattle examined, the parasite was only in 22 cases found to be spread through the whole muscular apparatus of the body. Zenker and Railliet have also discovered it in the goat.

The development of the parasite in cattle is essentially the same as in swine. But microscopical examination shows that it has no rostellum and no circlet of hooks, hence its name of *tænia inermis*, or unarmed tape-worm. But the four suction-cups are strongly developed, and on the crown of the head is also a frontal suction-disc. Hertwig's tests show that its organic development is ended in about 18 weeks, and its growth in 28 or a little more. Here also defunct parasites are found alongside the living ones. In 30 days they are  $\frac{1}{2}$  inch long and  $\frac{1}{4}$  inch broad, and the greatest length they attain is  $\frac{1}{2}$  inch;

The tape-worm whence these spring is much commoner in man than that which causes swine-measles, the latter having been almost extirpated in large towns by strict supervision and wise hygienic measures. But improved methods of examination have shown the cattle-parasite to be much commoner than was believed. According to Alix, one-fifth of the cattle slaughtered in Tunisia show its presence, which he explains on the ground that the beasts are only half fed and therefore consume dung-smeared fodder which better fed cattle would despise, and thus become infected with the eggs of the human tape-worm;

**Symptoms.**—The disease occurs in cattle without any symptoms. Even in the special feeding experiments no special signs were noticeable in many cases (Gerlach, Perroncito, Püts). But Leuckart, Mosler and Zürn, on the contrary, succeeded in producing a decidedly morbid aspect in a part of their trials on calves, which they dubbed with the very unfortunately chosen name of "acute cestoid tuberculosis," because of its peculiar sectional result, which reminded of tuberculosis. These phenomena consisted in rise of temperature, diminished appetite, signs of pain on palpation of the hind quarters, groaning, staggering, stiff and painful gait, increasing emaciation and debility, inability to rise up, constant lying, difficult breathing, diarrhoea, fall of bodily temperature and death on the twenty-third day. On dissection, thousands of the parasites were found in the heart, midriff and in the muscles of jaws, tongue, neck and belly.

**Diagnosis.**—It is usually impossible to recognise the measles of cattle during life, unless the presence of the parasite should be accidentally discovered under the tongue. It is said to be the rule in Southern France to examine the tongues of slaughtered cattle, as well as of pigs, for this purpose. But this invasion of the tongue by the parasite may lead to a confusion with antinomycosis, as Noack has pointed out. Moreover, its discovery is much more difficult than is the case in pigs, and probably it has hitherto been generally overlooked. Certainly the frequency of *Tænia mediocanellata* in man is out of all proportion to the discovery of cattle-measles in the slaughter-houses. This difficulty of demonstration arises partly from the fact that most of the parasites appear singly, and partly because, on contact of the flesh with air, they soon become invisible, and partly also because they often lodge in the

adipose tissue. The experience of the Berlin and Magdeburg abattoirs proves that their favourite haunts are the inner and outer muscles of mastication, of which the outer ones seem to be most frequently selected.

**Prophylaxis.**—As in pigs, this consists in hindering all consumption of the proglottides and eggs passed with human faeces, but is much more difficult. But at any rate, the staff employed in the byres should be prohibited from evacuating therein.

**Human infection** occurs solely through eating raw or half raw beef, especially under-done beefsteaks. The best protection then is thorough cooking quite through. The tape-worm of cattle-measles is much more difficult to drive out from the human body than *taenia solium*. As regards meat-inspection, measly beef should be treated exactly the same as pork which is infected. In Prussia, if a single parasite be discovered, and that only in the jaw-muscles, the whole carcase is condemned as injurious and may only be sold for domestic use after being boiled under police supervision.

### III.—MEASLES IN DOGS.

In isolated cases the larva of *taenia solium* has been found in the brain, muscles, liver, lungs, etc., of dogs, and the infection must have come from man. Usually the symptoms were those of cerebral affection : mania, epileptiform spasms, rotary movements and singularly sudden death. More rarely there were signs of muscular disorder. Thus in one case Trasböt noticed absolute incapacity for movement, with great pain when touched or when the limbs were stirred. Noack asserts that he also found the *cysticercus tenuicollis* in the brain of a dog. Finally, *cystice cus cellulose* has been discovered in sheep, cats, deer, bears and rats.

## CHAPTER XIV.

### MIESCHER'S TUBES OR RAINEY'S CORPUSCLES.

(*Sarco-sporidiae, Psoro-spermia.*)

**Natural History Notes.**—Our knowledge even to-day is very insufficient respecting the nature of the tubes discovered in 1843 by Miescher in the muscles, and which were to some extent re-discovered by Hesaling in 1854 and by Rainey in 1857. These Miescher's tubes are formations of spindle, roller or thread-shape in the interior of the primitive fasciculi of the muscles, whereby the latter are made to bulge out. They are microscopically small, sometimes visible as fine, white, elongated lines, especially in pigs; and sometimes they attain a length of  $\frac{1}{4}$  inch, as may be seen in the larger animals. They are mostly disseminated, although present in very large numbers, but sometimes agglomerations are found as large as a pea or a hazel-nut. They are externally bounded by a thick cuticula. Within the tubes are an extraordinary number of minute objects (germs), shaped like sickles, kidneys or beans, which lie together in a homogeneous fundamental substance grouped into divisions like the meshes of a net, by which the larger tubes are constricted like pouches. On puncturing the tubes, a milky, purulent mass oozes forth, containing millions of sickle-shaped germs, which may often be entirely squeezed out, so that only the empty husk remains.

These objects were at first regarded as fungi, then as accumulations of lymph-cells and then as a stage in the development of swine-measles; but are now shown by later investigation to be sporozoa, which live as parasitic colonies surrounded by a tube in the diagonally striated muscles (so-called sarco-sporidiae). So long as the tubes remain closed, they appear to be harmless. But if these burst, then the sickle-shaped spores within are changed into amoeboid migratory cells, which penetrate the

neighbouring muscles as an inflammatory infiltration (myositis sarco-sporidica s. gregarinosa) and produce a specific muscular disease with atrophy of the muscle cells and formation of tumescent inflammation-foci (white or grey sarco-sporidial tumours in the shoulder and loin muscles of horses, which are hard and as large as the fist). In sheep the early rupture of these tubes causes roundish, encapsulated tumours the size of a pea, which penetrate towards the free upper surface of the pharynx and pleura (sarco-sporidial cysts).

The development above described has been demonstrated chiefly by Pfeiffer. He considers the antinomycos-foci described by Hertwig and Dunker to be the same as these.

Blanchard has set forth the following system : Class : Sporozoa ; Order : Sarco-sporidiae. I. Family mischeridæ, parasites in the diagonally striated muscles with two varieties : 1st—genus mischeria (thin membrane, homogeneous); and—genus sarco-cystis (thick membrane, permeated by fine caniculi). II. Family balbianidæ, parasitic in the connective tissue (thin outer membrane, homogeneous), with one species : genus balbiania.

To this genus balbiania Rieck assigns the peoro-spermian pouches of the ruminants. These occur oftenest in the interstitial connective tissue of the pharyngeal muscular apparatus of sheep and goats. (Morat found 30.2 per cent. out of 900 pigs to have peoro-spermian nodules in the pharynx.) The genus mischeria is commonest in our domestic animals, but only occasional in pigs. Ripping asserts that Miescher's tubes were not absent from any pigs examined by him. Kühn noted them in 98.5 per cent. of all animals he examined ; and Koch in 8 per cent. of all pigs slaughtered in Vienna. Next in frequency of attack come horses and cattle. Rieck states, as the result of his observations, that although the sarco-sporidiae do not, as a rule, cause any marked pathological changes in the muscles, yet that under some circumstances they may produce an interstitial myositis, at first acute and then chronic, with secondary degeneration of the muscular fibre. The former arises from the immigration of the sarco-sporidial germs into the interstitial connective tissue of the muscle. With the penetration of the germ-like formations of development of the sarco-sporidiae into the muscular fibre and their encysting there, the second or chronic stage of the inflammation begins, whereby a transformation takes place of the cellular exudation into fibrillar connective tissue. The opinion expressed by Siedamgrotzky, Laulané and Brouwier that the chronic interstitial muscular inflammation occurring in horses, cattle and pigs is caused by the sarco-sporidiae found therein, is very much strengthened by these observations of Pfeiffer, as opposed to the contrary opinion expressed by Pütz, Eberth and Schmidt-Mülheim.

**Occurrence.**—Miescher's tubes are only found in the muscles of herbivora and omnivora, especially in pigs, the ruminants (ox, sheep, deer) and in horses. They have also been

met with in buffaloes, reindeer, goats, hares, rats and mice, in fowls and other birds, but never yet in the carnivora. The largest tubes (*sarco-sporidiae gigantea*) occur in goats, sheep and buffaloes. They are most frequently found in the muscles around the cavities of the mouth and throat—for instance, in the oesophagus, tongue and muscles of the jaws, cheeks, larynx and pharynx; although frequently numerous in other muscles, such as the neck, loins, abdominal walls, thighs, midriff, heart, etc. Sometimes they lead to interstitial myositis with atrophy and cellular infiltration of the connective tissue of the surrounding muscular fibres. In other cases the flesh—e.g., that of full-grown cattle—assumes a pale colour like that of veal, owing to the presence in large quantities of *sarco-sporidiae*. In older horses and in buffaloes they seem to be almost invariably found; also in pigs Perroncito discovered them in a quarter of those examined. Among 100 cachectic sheep examined by Moulé, 99 had *sarco-sporidiae*.

**Symptoms.**—Frequently as Miescher's tubes occur in the muscles of herbivorous domestic animals, they rarely cause any signs of illness in their hosts; only in solitary cases have any pathological symptoms been observed which arose from muscular changes of inflammatory nature and were caused by an excessive number of these tubes. Thus Dammann and von Niederhäusern noticed difficulty of breathing in a sheep and a goat caused by inflammation in the muscles of the larynx and pharynx; Brouwier and Tokarenko observed lameness in a bull and difficulty in rising, and, indeed, complete muscular paralysis; Virchow, paralysis of the hinder limbs in pigs. Brchoenionski noticed loss of appetite in two pigs, continual lying, arching of the back, irregular movement of the hinder parts, pain in the muscles when pressed, hoarseness and fever (trichinosis at first suspected); after slaughtering, the muscles were found to be watery, opaque and permeated by numerous *sarco-sporidiae*.

Anatomical changes are more frequently found in the flesh of slaughtered animals. According to Hertwig, Miescher's tubes were so enormously numerous in six pigs that the muscular preparations contained as much of tubular as of fibrous matter. The flesh was consequently flabby, extremely watery, and its section-surface assumed after a few hours a pale green colour. (For differential diagnosis, consult the chapter on Trichinosis.)

## CHAPTER XV.

### DISEASES OF THE NERVOUS SYSTEM.

#### I.—DISEASES OF THE BRAIN AND ITS MEMBRANES.

*Inflammation of the Brain. Meningitis and Encephalitis.*

**General Notes on Inflammation of the Brain.**—The classification of the various forms of brain inflammation occurring among domestic animals varies as we regard them from the anatomical or the clinical standpoint. Viewed from the former we distinguish three kinds, viz. :

1. *Pachy-meningitis, or inflammation of the dura mater.*
2. *Lepto-meningitis, inflammation of the pia mater.*
3. *Encephalitis, or inflammation of the cerebral substance itself, including brain-abscesses and also some softenings of the brain.*

Lepto-meningitis may be either serous, suppurative, croupous or tuberculous in type. We also speak of a convexity-meningitis and a basilar-meningitis, according to the seat of the inflammation. A simultaneous affection of the meninges and of the brain is named an encephalo-meningitis. But this exact, anatomical classification, excellent as it is, cannot be adopted in a clinical description of these various diseases in domestic animals. The conditions are very different from those of human pathology. First of all, a clinical differentiation between meningitis and encephalitis in domestic animals is extraordinarily difficult, if not impossible, as they present essentially the same symptoms, and because when the membranes are inflamed the brain itself is generally also implicated. It is certain that what is classed as inflammation of the brain in our veterinary medicine is in far the greater number of cases lepto-meningitis. Positive proof, however, in individual cases can only be obtained by dissection. To diagnose a specific form of lepto-meningitis during life is also very difficult, except, perhaps, in the tuber-

culous basilar meningitis which occurs in cattle (compare tuberculosis). In this connection we can only add that simple serous basilar lepto-meningitis is the commonest form of meningitis, whereas the suppurative or fibrinous seems to be much rarer. Relatively easier appears to be the diagnosis of a pachy-meningitis, when we have to deal with injuries to the skull, diseases of the ear, etc.; and yet here it can never with certainty be decided in life how far the irritation has extended from the bony covering to the softer membranes and brain itself. Definite clinical proof of encephalitis can only be established with certainty when being circumscribed, it presents distinctly central symptoms, or when the metastatic development of a cerebral abscess may be presumed. But here, also, we must observe that central or special sense symptoms may be noticed in a number of other brain diseases (haemorrhage, tumours), and that a suppurative meningitis may metastatically develop instead of a cerebral abscess. These circumstances will explain why we do not here adopt that classification of the different forms of brain-inflammation which we are bound to acknowledge as the best, because most exact, both pathologically and anatomically.

The clinical sub-division of brain-inflammations hitherto found most serviceable was into the acute and sub-acute forms. Under the former were included all rapid cases with extreme phenomena of cerebral inflammation (excitement), and under the second those which presented signs of depression and were also slower in their course. This differentiation is, however, only one of degree and by no means essential. Then, too, every meningitis or encephalitis can, under given circumstances, occur either as acute or sub-acute. Moreover, experience shows that transitions and intermediate stages between the two forms may arise, so that no strict boundary can be set between them. Finally, the morbid aspects of acute or sub-acute inflammation of the brain furnish no clue to any uniform process within the brain. The following brain diseases may occur with the aspect of "acute inflammation of the brain," e.g., hyperæmia of the brain, pachy-meningitis, lepto-meningitis (serous, purulent and tuberculous), encephalitis, emboli and, under some circumstances, also tumours in the brain. But the morbid aspects of "sub-acute inflammation of the brain" are much more comprehensive, as, besides the cerebral affections just named, other conditions are also included therein, which shall be more fully specified in speaking of the sub-acute brain inflammation

of horses. For these reasons we have not been able to decide in favour of this method of sub-division.

I.—INFLAMMATION OF THE BRAIN IN HORSES. LEPTO-MENINGITIS, PACHY-MENINGITIS, ENCEPHALITIS.

**General Observations on the Definition of Sub-acute Encephalitis in Horses.**—In contrast to the "acute" form in horses which is accompanied by great excitement, a second, or "sub-acute," form has been recognised, which manifests itself chiefly by symptoms of brain pressure and stupor. The connection of the two has been indicated by the descriptions: "Inflammation of the brain with mania" on the one hand, and "inflammation of the brain with stupor" on the other. Other synonyms for sub-acute brain inflammation are: Inflammation of the brain with somnolence, hot head-disease, acute staggers of asthenic character, acute hydrocephalus, semi-acute inflammation of the brain, semi-acute head-disease, passive inflammation of the brain, etc. The multiplicity of these names shows that the "sub-acute" form of the disease is more important than the "acute," and we therefore add a few general observations concerning it.

That it is no uniform disease in horses is plain both from veterinary literature and from the results of dissection. Many practitioners still give this name to all acute or inflammatory brain-affections, the following of which seem rightly to belong to this category:

1. *Lepto-meningitis serosa*, which is probably in its essential a lepto-meningitis basilaris. This form may occur either acutely with symptoms of excitement, or sub-acutely with those of depression. It obviously represents the principal variety both in "acute" and "sub-acute" inflammations of the brain.

2. *Various other brain diseases, both inflammatory and non-inflammatory* in their nature, exhibit also the aspect of a "sub-acute" brain inflammation, and are therefore probably often included under this title. To these belong *encephalitis, pachymeningitis, hyperæmia of the brain, hemorrhage on the brain and tumours of the brain*.

3. *Poisoning by food-stuffs* seems also to have been regarded as inflammation of the brain whenever any specific brain-poison has been generated, perhaps by the attacks of fungi. Here especially lupins, clover (compare lupinosis and clover-sickness, Vol. II., p. 199) and leguminous foods must be cited. Also

in a few cases the action of narcotics or acrid narcotic plants (*equisetum*, bearded darnel, yew-tree branches, etc.). The very frequent attachment in veterinary literature of blame to leguminous plants (to clovers, vetches, etc.), as well as the precedent gastric symptoms mentioned therein (colic, loss of appetite, icterus), all point to the connection between "sub-acute" brain-inflammation and poisoning by food-stuffs.

4. Finally, the phenomena sometimes remind us of an *infectious disease*. The frequent simultaneous affection of several animals, and the suggestion so often made that foul, close stables have been primarily responsible for the outbreak, and above all the absence of any inflammatory changes in the brain or in its membranes on *post mortem*, all produce the impression that we are dealing with a primary, general disorder of the blood, followed secondarily by cerebral symptoms, and therefore resembling an independent brain-affection. In addition to this, dissection proves the existence of other organic diseases altogether characteristic of infectious maladies, such as opaque swelling or fatty degeneration of the liver, decided decomposition of the blood or the formation of ecchymoses, and the picture is complete. As regards the nature of the poison, whether it be chemical or bacterial, whether absorbed from without or generated within (auto-infection), nothing fuller is known. Some assume a peptone-poisoning of the body as causing the "sub-acute" brain-inflammation, and originating in a transformation of the albuminous corpuscles of the blood by the ferment contained in leguminous food. Although this assumption is nothing more than a hypothesis and must be restricted to individual cases, yet it indicates the necessity of a decomposition of the blood in order to account for the disease.

**Etiology.**—As causes of cerebral inflammation the following factors must be especially noted :—1. The prolonged maintenance of *too high a temperature* in steamy and badly ventilated stables, keeping horses in very warm cattle-byres, abnormally warm weather, sultriness. 2. *Sudden changes in external conditions*, such as change of owner, transport by rail or ship, removal from elevated to low-lying districts, drafting into cavalry, or being placed in an exhibition. Cases have been known of horses being affected by brain-inflammation several times in one to two years, and every time they changed owners. Among cavalry remounts and market horses other causes besides the frequent change of surroundings may also contribute, such as over-

feeding, over-exertion, chill, bad stabling, etc. 3. *Over-exertion*, especially after long resting in the stable and too high feeding; the disease is then not rarely observed in the same stables in which cases of haemoglobinæmia simultaneously occur. 4. *Too rich feeding* has always been charged with being a cause, and especially with leguminous fodder (peas, beans, vetch, clover), also with food which is too fattening and too nitrogenous, particularly certain kinds of grain, e.g., summer rye. In fact, the opinion was formerly held that the occurrence of the disease is exactly proportioned to the leguminous harvest of the district, and that it is unknown where such crops are not grown; but this last assertion has been proved untrue. It must, however, be admitted that food which is too rich and nitrogenous produces a predisposition to inflammation of the brain and may be classed as one of its causes. 5. *Injury to the roof of the skull*, even simple contusions, also suppurative inflammation of the cranial-bones, especially the temporal bone (suppurative inflammation of the middle ear); serious operations within the cavities of the nose, forehead or upper jaw produce first pachy-meningitis, upon which both lepto-meningitis and encephalitis may supervene. 6. *Parasites, new growths, emboli and thromboses* in the brain may also give rise to brain-inflammation. Among parasites the larvae of oestrus occur, although rarely, in the brain, and produce general phenomena of brain-irritation or interference with special senses; then also the larvae of *struthionis armatus*, and finally, coenurus-vesicles and cysticercæ (compare *stammers*, Vol. II., p. 590). Of new growths we must mention especially cholesteatomata, which, when very large, may very suddenly cause acute inflammation of the brain without any premonitory signs having been observed. The same applies to sarcomata of the ethmoid bone. Emboli are most commonly noticed during glanders and pharyngitis; they usually cause circumscribed brain-abscesses. The formation of thrombosis in the cerebral arteries is also known to provoke an inflammation of the brain. 7. It is also secondarily produced by various general disorders and especially by *infectious disease*. Besides strangles and pharyngitis, which may develop by means of emboli into a suppurative encephalitis, septicaemia and pyæmia are particularly liable to be complicated with suppurative lepto-meningitis; thus, for instance, in the "joint ill of foals." Brain-inflammation is also noticed in the course of pleuro-pneumonia and variolous fever.

Horses not yet fully developed show a predisposition to the

disease, for which reason its occurrence is most frequent between their second and sixth years. But even very old and quite young animals may also be attacked. Horses, moreover, descended from sires or dams which have previously suffered from inflammation of the brain or staggers show a decided liability to the complaint. Definite data respecting the influence of race are not yet to hand, but languid, common country horses seem to be more readily affected than those of nobler breed, though the complaint is moderately common in these. Military horses, though not quite exempt, as some have affirmed, are yet but seldom attacked, which is probably due to their regular feed and keep. As regards seasonal influences, more cases occur in spring and the early half of summer, but they take place all the year round. In some districts the malady seems to be almost enzoötic, and in certain stables even permanent.

**Pathological Anatomy.**—1. **Pachy-meningitis** when *acute* is usually suppurative. The dura mater is found to be hyperæmic, infiltrated with haemorrhages, its inner surface opaque and covered with fibrinous or purulent deposits; sometimes the dura has a fleshy appearance. **Chronic pachy-meningitis** is characterised by fibrous proliferations of connective tissue, which result in adhesion of the dura to the roof of the skull; at the same time, bony new-growth takes place (*P. ossificans*).

2. **Lepto-meningitis (serous)** begins with hyperæmia of the vessels of the pia, and swelling, opacity and a spotted or striped haemorrhagic reddening of the soft cerebral membrane. Between the pia and arachnoid or dura we find an accumulation of yellowish-red fluid (*hydrocephalus externus*), which, if abundantly present, flattens the cerebral convolutions. The brain-substance directly beneath the pia is oedematous, its sectional surface showing a moist lustre, and, if it be pressed, a little serum gathers later in the hollow thus made. This oedematous saturation may develop into complete softening, when the brain-matter takes on a pulpy consistence; occasionally haemorrhage is found in the brain. In the ventricles serum has accumulated, usually bright and clear, but sometimes dull and bloody, and varying much in quantity (the average is 1 to  $1\frac{1}{2}$  oz., but sometimes only a few drams). According to Schütz, out of twenty-five cases of acute hydrocephalus he found in the lateral ventricles from  $\frac{1}{2}$  oz. to  $1\frac{1}{2}$  oz. in twenty-two instances, once  $1\frac{1}{2}$  oz., once 2 oz., and in one case  $2\frac{1}{2}$  oz. of the fluid. When

there is much of this, the optic thalamus and the corpus striatum are flattened and the ventricles dilated; the cerebral substance around the ventricles is oedematous and softened. The network of veins is also oedematous, swollen, and forms flabby, greyish-yellow and puffy lumps.

*Suppurative lepto-meningitis* is usually a convexity-meningitis. In addition to hyperæmia we find haemorrhagic redness and swelling of the pia between this latter and the arachnoid; also between arachnoid and dura greyish-yellow, flaky or moderately firm exudation, which consists mainly of pus-corpuscles and finely granular fibrin, and is also croupous in its nature. The next adjoining portion of the cerebral cortex is sometimes hyperæmic, sometimes anaemic and oedematous, or even purulently infiltrated (meningo-encephalitis). The ventricles of the brain are usually empty.

*Chronic lepto-meningitis* is manifested by adhesion of the arachnoid to the dura, by milky opacity and thickening as well as internal adhesion of the pia to the brain-substance, into which it projects strong processes of connective tissue. The veins are dilated and their walls thickened; sometimes both chronic oedema of the pia mater and serous effusion and atrophy of the upper surface of the brain are visible. Also in the ventricles one may occasionally find small quantities of fluid, considerably dilated vessels of the ependyma with thickening of the membrane itself.

As regards the accumulation of serous exudation in the ventricles during lepto-meningitis, we must remark that its quantity is very variable. Even in sound horses the amount of cerebral and cerebro-spinal fluid varies considerably, but much more in those that are ill. Thus in four horses with brain-disease, the total quantity of the cerebro-spinal liquid was from 4 to 9 oz. (the cerebral alone being 1½-4 oz.), while in three otherwise diseased horses it reached from 10 to 13½ oz. Hering concludes rightly from this that so-called "brain-pressure" cannot be the cause of the morbid symptoms in inflammation of the brain. Moreover, we often meet with false views as to the connection between the ventricles of the brain and the sub-arachnoideal cavities. There is no direct relation between them. It is also impossible for the fluid to escape from the ventricles on decapitation. The amount, therefore, found in the lateral ventricles on dissection agrees exactly with that which was present in life. The sub-arachnoideal fluid which escapes when the head is cut off is usually mistaken for the exudation in the ventricles.

3. *Encephalitis* is generally partial or circumscribed; only rarely is it general or diffuse.

(a) The *circumscribed non-suppurative* form presents irregular round foci, which are usually from the size of a pea to a hen's egg, but may also embrace an entire cerebral lobe and are not sharply defined from their surroundings. At first the places in question are almost imperceptibly diffusely or distinctly spotted with red, owing to hyperæmia or haemorrhage. To this are soon added, as a result of serous exudation, swelling and softening of the brain-substance, when the microscope shows the cells of the neurolia (neuroglia), as well as the ganglionic cells to be swollen, granular opaque and in a condition of fatty degeneration, the axis-cylinders irregularly changed and the tissue of the glia infiltrated with small cells. The encephalitic focus undergoes maceration, swelling and breaking-down, and finally represents a softened substance consisting of decomposed and fatty-degenerated glia and ganglion-cells, white blood-corpuscles and free nodules of fatty granules, and is then described as a simple brain-inflammation or inflammatory softening of the brain. This may be distinguished from the non-inflammatory form, caused by disturbed nutrition, by the infiltration of white blood-corpuscles, which is its characteristic sign. At this stage the inflammatory process may terminate. But it is very often complicated with haemorrhage, as the vessels traversing the encephalitic focus may undergo fatty-degeneration and rupture. This condition is known as haemorrhagic encephalitis, or inflammatory red softening of the brain. As the colouring matter of the blood becomes decomposed in the above-named focus, it gradually loses colour and appears yellowish—this is inflammatory yellow softening of the brain. The decomposed and exuded matter may liquefy and be re-absorbed, forming then either a grey, gelatinous focus of disintegration (grey inflammatory softening) or a cyst, in some cases, a sclerosis of connective tissue, or a cicatrix.

(b) *Partial suppurative encephalitis* appears as an abscess in the brain. It develops especially after septic or pyæmic emboli from haemorrhagic inflammation centres, which gradually enlarge and undergo purulent disintegration. A granulating membrane may form around the abscess, which shuts in the pus-cavity. This pus is at times yellowish, greenish or discoloured; its consistence is that of cream. Such abscesses of the brain are most frequently observed in strangles of horses, and also in cattle. Usually there is only one abscess, but occasionally several are found, and in very rare cases quite a large number in the same animal;

(c) *Diffuse encephalitis* is very rare. Friedberger observed in one horse, which during life showed the customary signs of inflammation of the brain, no changes in the brain beyond a diaphanous, yellowish colour like amber spread over the white matter of both frontal lobes of the large cerebral hemispheres. The microscope showed that the peri-vascular cavities as well as the neuroglia were closely permeated by white blood-corpuscles; the capillaries appeared hyperæmic. No other changes could be perceived.

**Symptoms of Inflammation of the Brain in Horses.**—The phenomena of this malady in horses are exceedingly variable, sometimes the signs of excitement predominating and sometimes those of depression, while very frequently the two are commingled. Both the degree, form and seat of the inflammation cause great differences, as do also the temperament and intelligence of the affected animal. For these reasons no typical picture of inflammation of the brain can be drawn, and we must restrict ourselves to a delineation of the several symptoms.

1. *Signs of excitement* generally open the scene in the severer cases, but they may be absent, or not appear until later. They consist in restlessness and excitement, which usually appear unexpectedly and suddenly in the stall, or when being ridden or driven, and may amount to raging mania. The horse presses or leaps forward or sideways, rears up, hangs upon the halter or snaps it, tumbles down, rolls over or strikes its head against the wall, by which means it may receive serious injury both of head, breast and limbs. It can no longer be led, or only with difficulty, has a hasty, thrusting, groping movement, runs at every obstacle and, if once taken out of the stable, can only with much trouble be got back again. The eye is wild or staring and its pupil contracted. The papilla of the optic nerve is found, on examination by the ophthalmoscope, to be redder and its vessels more highly charged. Sensibility is heightened; a touch upon the head or ears, a word spoken, or even the slightest sound, increase the attacks. Muscular spasms are also observed, especially in the muscles of the face, and show as twitching and tremor of the lips, or as spasms of the jaw (grinding the teeth); also in the muscles of the neck and extremities and at times also of the diaphragm (sobbing). Stallions often project their penis and show erection.

2. *Symptoms of depression* occur either in connection with

those of excitement, or at the commencement of the outbreak ; but in extreme cases which rapidly end in death they may be absent. Sometimes they appear suddenly, at others after certain premonitions, such as lassitude, laxness or loss of appetite, and reveal themselves chiefly in a sleepy, unconscious condition. The head is hung down or rested on the crib, the animal leans against the stall, its eyes closed as in sleep, with the pupil dilated, and will remain thus for hours, or for half a day ; or it may adopt unnatural postures, crossing its legs, or drawing them all into a bunch, or perhaps straddling them wide apart. Sometimes it loses its balance and topples over, or doubles up its joints, or stumbles forward, especially when trying to pick up food. It cannot be roused from this sleepy state, or only for a short time and by dint of much shouting. Only rarely does the horse lie down, and then can only with difficulty be made to rise again, when it sometimes remains a long time on its knees. Its gait is uncertain, stumbling and tottering, the legs being either lifted too high or not high enough ; and rotary movements are often observed, lasting even for hours and mostly in one direction. Sensitiveness may also be much diminished, and one may tread on the coronet joint, pull the ears, clap the hands before the animal's face or strike it with the whip without eliciting any sign. Finally, symptoms of paralysis may develop, e.g., paralysis of the retina or centre of vision (amaurosis) of the ear, pharynx, upper eyelid (*ptosis*), hind-quarters ; and also, in encephalitis, of the muscles of one side or half the side.

3. *Fever* is usually present and may rise to 104-106° Fahr., shivering fits may also be observed, especially in suppurative meningitis and encephalitis. The fever is in general regulated by the intensity of the irritation manifested. But it is often entirely absent when depression predominates, and even in cases of extreme excitement the temperature may continue quite normal. The pulse is very full and accelerated in the stage of excitement, but grows feebler and slower afterwards, and may even sink below the normal point (to 24 to 26 strokes a minute), but it is very easily affected, so that its frequency may vary many times during a day. Towards the fatal end its speed is extremely rapid. External bodily temperature is mostly irregularly distributed ; at the outset the mucous membranes of the head are very red ; and sometimes, though by no means always, the papillæ of the optic nerve are hyperæmic.

4. *Appetite* is in severe cases *entirely suspended*, so that the

animals literally perish of hunger and thirst ; in lighter cases it is much diminished. Eating is done irregularly and contrary to physiological methods, bad food being very often preferred to good, or it is taken hastily, snatched, and large wisps of hay or straw are seized, then partly chewed and carried a long time in the mouth. The animal always prefers to feed from the ground. As a consequence of paralysis of the pharyngeal muscles, portions of food may stray into the larynx, causing pneumonia by the introduction of foreign bodies. Peristalsis is usually delayed and also evacuation and urination. Exploration of the rectum often finds it charged with masses of faeces.

5. *The breathing* is also accelerated during excitement, but later the number of respirations often sinks below normal, and may be as few as six per minute ; but they are very deep and slow, and each inspiration is sometimes marked by a snoring sound. Whenever the breathing is very rapid during the prevalence of depressional symptoms, this indicates the existence of pneumonia, and the lungs must be carefully examined.

**Course.**—Inflammation of the brain may run a very acute course and very soon end fatally, sometimes even within the first twenty-four hours, by apoplexy. At other times the result is not seen for many days or weeks (acute and sub-acute course). The former begins either with uninterrupted mania, followed by continued somnolence, or the maniacal symptoms appear in outbursts with intermissions, and, finally, the state of depression may also be several times interrupted by signs of excitement. Only in very slight cases does recovery follow in two to four weeks ; in severer ones death usually occurs within the first fourteen days, or else the case turns to sleepy staggers (chronic dropsy of the ventricles). Improvement and relapse very frequently alternate. Weather has considerable influence upon the course of the disease, great heat being very unfavourable, while cool nights, rain, etc., have a very good effect.

**Prognosis.**—The forecast in brain inflammation is somewhat unfavourable. The average number of cases of complete cure is only 20 to 25 per cent. The proportion of those which pass into the chronic form of hydrocephalus or sleepy staggers must be set at least as high, or even higher, than that of the cases ending fatally. Among other after-consequences must also be named : vertigo, general muscular weakness, epilepsy,

amaurosis, deafness and also monoplegic and hemiplegic paralyses; moreover, a recovery from the disease always leaves a disposition to relapse. Severe cases almost invariably end fatally. But that there are exceptions to this rule is shown by Hering and others, who observed recovery in the fourth week, after the animal had lain for days on the ground in a hopeless state. The cause of death is either paralysis of the brain, pneumonia due to some foreign body, or perhaps decubital gangrene with septicæmia or pyæmia, and sometimes hunger and thirst.

**Differential Diagnosis.**—Inflammation of the brain may be mistaken, on the one hand, for hyperæmia and mania, and on the other for sleepy staggers. (Compare the differential diagnosis of these two complaints.) Certain poisonings may also lead to confusion, such as those from beech-nuts and yew-tree sprouts. Autopsy is generally the only sure guide.

**Treatment.**—The first thing to be done with a horse suffering from inflammation of the brain is to bring it into a *cool and quiet place*, which is as airy and dark as possible, and where it can move freely about without injury to itself or others. Loose boxes are therefore best, or, if not available, then a barn or shed plentifully strewn with straw, or a fenced paddock. In the latter one may leave the patient over night, if the weather be not too severe. The disease must then be attacked both locally and generally. To the immediate neighbourhood of the brain apply ice, snow or cold-water bandages, or, if necessary, a so-called cooling mixture of saltpetre, common salt and glauber salt with vinegar or water, and continue these as long as the signs of abnormal flow of blood to the brain are maintained. Later on, when symptoms of stupor predominate, pouring cold water on the head is better; this should be done with a watering-can and from different heights, as this has also a stimulating effect. These cold bandages should be helped by cold infusions of the rectum, whenever this can be done. The practice of blood-letting formerly adopted in all cases of brain-inflammation is only permissible at the outset of the disease, or when pronounced symptoms of active hyperæmia of the brain are present. In all other cases it must be avoided, because, if exudation have already taken place in the brain, it only increases the anæmia of the brain caused by pressure of this exudation, and thus exacerbates the disease.

*Derivation to the bowels* is also prescribed. But here, as in hyperæmia of the brain, we can only recommend the true drastics (croton oil, aloes, tartar emetic) for the beginning of the malady, and only when real hyperæmia to the brain is still present, and the animal's constitution permits their use. In other instances drastic remedies, according to our experience, are just as injurious as bleeding, as by diverting the blood too much to the intestines they only increase the anæmia and depression of the brain. On the other hand, gentler purgatives (neutral salts, or smaller doses of calomel) are often useful, especially if the bowels be obstructed, and even if depression have already set in; that is to say, if it be found possible to introduce them by the mouth. If this cannot be done, then recourse must be had to subcutaneous injections of physostigmine ( $1\frac{1}{2}$  grains per dose), or arecoline ( $1\frac{1}{2}$  grains).

Attempts are also made to cause resorption of the fluid which has collected in the ventricles and between the membranes of the brain, by administration of pilocarpine, which is usually injected for several successive days in doses of three to eight grains. Although it must be granted that pilocarpine renders good service in many cases of acute and sub-acute hydrocephalus, yet experience has shown that it is not an infallible remedy, but often enough leaves the practitioner in the lurch, or even makes the case worse. We should also like to advise caution against using it in too large doses. The same caution applies to arecoline ( $1\frac{1}{2}$  grains).

*Derivation to the skin* by sharp inunctions of croton oil, mustard oil or cantharides-ointment near the neck or on both sides of the throat are objectionable during the stage of excitement, as they only increase it. Their use in conditions of stupor is also doubtful, so far as our own experience goes; the same applies also to the cautery and setons. Others, on the contrary, highly recommend inunctions and derivations. For conditions of extreme excitement the administration of bromide of potassium or chloral hydrate 6 to 12 drams (rectal) is to be recommended. The sick animals must not have too rich or too stimulative food; as a rule we give them no oats, but bran, meal-drinks, green fodder, turnips, etc. Prophylaxis consists in avoidance of the causes already mentioned; for full-blooded and well-nourished horses the occasional administration of a purgative is advisable, or a daily dose (6 to 12 drams) of artificial Carlsbad salt.

II.—*INFLAMMATION OF THE BRAIN IN OTHER DOMESTIC ANIMALS.*

This disease offers in other animals the same general conditions in regard to etiology, pathological anatomy and therapeutics as in horses, yet the malady varies somewhat in different animals, so that its symptoms must be briefly discussed.

In cattle the excitement manifests itself in bellowing, snorting, shaking the head, thrusting with the horns, which may even be broken off, gaping, foaming, leaping into the manger, charging and overthrowing objects lying in the path, trembling, epileptiform spasms, crib-biting and gnawing, a glaring look in the eyes, rolling the eyes, spasmodyc uplifting of the legs, rotary movements, butting forwards and striking the head against the wall. Later on we find stupor, a groping gait, unphysiological attitudes, lessened sensibility, collapse, general signs of paralysis and amaurosis. The course is generally very acute, and the condition generally ends in death within a few hours or days. Confusion may occur especially with malignant catarrhal fever (head-sickness of cattle); but differentiation is generally possible by noting the affections of the eyes and of the nasal and frontal cavities. Acute lead-poisoning and poisoning by corn-poppies may also present similar symptoms.

2. In dogs brain inflammation generally exhibits premonitory signs, which are disquiet, timidity and a desire to hide. The dogs no longer know their masters, sometimes they howl continuously, or scream if touched. Only rarely do they bite, and only when seized; indeed, aggressive conduct towards man or other animals has never been observed. Fits of mania are very rare, and seem only to arise when the dog is fastened into its cage, when it will bite at the bars and tear up its bed. In the open it runs aimlessly about and knocks against things so violently as to be thrown down. Its glance is wandering, the conjunctive are very red, the skull feels very hot, the pupils are contracted; vomiting also and staggering, epileptiform spasms and even involuntary movements are noticed. Later the dog lies paralysed, stupefied and unconscious. The result is often very quickly fatal; but chronic conditions of depression may also develop, viz., hydrocephalus, amaurosis, deafness, etc. Inflammation of the brain is much rarer among

dogs than horses, the statistics of the Berlin dog-hospital showing only 103 cases out of 70,000 dogs treated.

Confusion may arise especially with rabies; and often only autopsy can decide the question. But in brain-inflammation we lack the aggressive action, the altered voice and the consumption of indigestible substances; while rabies does not present such intense injection of the conjunctivæ, or so high a temperature of the skull. A very similar aspect is also presented by nervous distemper, pentastoma tænioides, worms in the bowels and several forms of poisoning (filix and santonin).

3. **Pigs** show attacks of mania, climb up the walls of their sty, utter piercing cries, grind their teeth, dribble, fall into convulsions, have fits of twitching and epileptiform spasms, twist round and round, press hard against the side of the sty or other object, sometimes collapse entirely; their skulls and the root of their ears feeling very hot. Later they hang down their heads and show signs of stupor and paralysis. Pregnant sows abort. The end is frequently fatal, recovery being rare; but a condition of sleepy staggers may remain behind.

4. **Sheep** are said to hold their heads stiffly in one position, press forward, twist about, stagger and show twitchings. The complaint is not easy to distinguish from staggers (*cœnurus cerebralis*); in general, though, the course of the latter is more chronic.

5. **Goats** also hold the head and neck stiffly, grind their teeth and have epileptiform spasms. In sheep and goats it is not always easy to decide whether the symptoms arise from simple inflammation of the brain or from cerebro-spinal meningitis.

#### SLEEPY STAGGERS (*Chronic Hydrocephalus*).

**Occurrence.**—Chronic dropsy of the cerebral ventricles is most common in horses, in which it represents the chief cause of sleepy staggers; and is rarer in cattle, dogs, pigs and sheep. Among horses those of middle life are most subject, so that it rarely appears earlier than their fourth year; geldings, moreover, seem to be more prone to attack than stallions or mares. This is perhaps explicable by the deterrent effect

of castration upon brain development, an effect which has been scientifically demonstrated in several animals; and any organ which has been checked in development has naturally less power to resist external influences. It is also universally believed that common breeds, such as those from low-lying, often-flooded regions, with lax constitution, dull and phlegmatic temperament, broad heads yet narrow skulls, have a special disposition to chronic dropsy of the ventricles.

**Etiology.**—Its causes are: 1. previous inflammation of the brain, complicated either with acute or sub-acute hydrocephalus. As this becomes chronic, it gradually changes after the fourth week into sleepy staggers (so-called symptomatic staggers). 2. The causes of so-called idiopathic staggers, i.e., of a gradually and slowly developed staggers independent of the above-named factor, are not yet clearly known. Heredity is established as one of these, for experience has long since shown that the offspring of horses which have suffered from the disease are also frequently subject to it in their later years. Curdt even asserts that he witnessed a case in a colt only two days old. In France also, it is said that sleepy staggers was almost enzoötic in certain stables of the Alps and Rhone valley as a result of heredity, so that breeding had to be entirely relinquished. But whether the malady arise from disease of the venous net-work, from chronic hyperæmia, or from a permanently higher blood-pressure in the brain, cannot at present be decided. As accidental contributory causes repeated active and passive cerebral hyperæmia, along with great heat, over-exertion and over-feeding, particularly with cereals and leguminous food, have always been blamed, especially if there be also plethora, liver or other gastric affections, disorders of the heart and lungs which disturb circulation, or compression of the jugular vein by the harness, etc.

**Post-mortem Lesions.**—The anatomical changes in chronic dropsy of the brain-ventricles consist first in the collection of a serous fluid, as clear as water, in the lateral ventricles of the brain, and sometimes also in the third ventricle. These lateral ventricles are thereby more or less enlarged, and not rarely to a considerable extent, so that perforation of the dividing septum may occur, allowing free passage from one to the other. The optic thalamus and corpus striatum, as well as the cornu ammonis, become flattened by the pressure.

of this liquid (atrophy of the brain), and the olfactory bulbs are sometimes bulged out into thin sacs. The ependyma of the ventricle is thickened and made rough and uneven by deposits of connective-tissue granulations ; the adjoining brain-substance appears anaemic, pale in colour, dryer and harder. The venous plexus is thicker, gelatinous, swollen and shows dilated and sinuous vessels. The volume of the brain appears to be increased, its convolutions are flattened, the furrows smoothed out and the superficial cortex is dry and anaemic. If the cerebral mass be removed to the roof of the ventricle, the latter bulges upwards and, upon puncture, the contents squirt out in a stream. The occurrence of the morbid symptoms is easily explained by these anatomical changes ; on the one hand the pressure is exerted by this fluid matter, and on the other the partial decay of the brain-substance.

**Symptoms.**—The aspect presented by chronic hydrocephalus or sleepy staggers is chiefly a disturbance of consciousness, perception, volition and sensation, with unusual actions and movements on the part of the animal ; also secondary disturbance of the heart's action, of digestion and respiration —other conditions being normal and especially there being no fever. But the malady may occur in varying degrees and with all sorts of complication of the several symptoms. The most important signs in horses are :

1. *Disturbance of Consciousness*, or sense of individual existence. This is revealed by the eye and bodily attitudes. The former is often half shut, the upper eyelid being drooped, and the glance is frequently quite expressionless, fixed and dull ; the animal looks as though sleepy. The head is generally sunk, or else laid upon the bars, manger or knees, or perhaps leans against the wall. The position of the legs is exceedingly strange and unnatural ; sometimes they are withdrawn far under the body or even all to one point, at others, they are crossed, or are planted in front, or one upon the other. They can, moreover, be placed in these or other positions at will, and will often remain thus for a very long time. These disturbances of consciousness increase if the animal be moved. Horses afflicted with sleepy staggers frequently manifest a strange desire to thrust either to the right or left, and this dulness of the sensorium is always greatest immediately after exercise.

2. *Disturbances of Perception*, i.e., of its relation to the

outer world. The animal takes no notice of its surroundings, or gains false impressions of them, which facts are first shown by the erratic motion of its ears. This is variable and contradictory, being not towards a sound but away from it, and not seldom one ear listens backwards and the other forwards. But this disturbed perception betrays itself oftenest in the taking of food and drink. The fodder is first seized, perhaps, in large bunches, chewed for a while, and then allowed to rest quietly in the mouth, as though chewing were forgotten ; then it will be taken more slowly and fully masticated, though with frequent pauses. Generally, also, the horse feeds rather from the ground than from the rack, which may perhaps arise from the altered pressure of the fluid when the head is raised or sunk. Many horses when suffering from sleepy staggers stand too near the manger ; they pull hay from the rack by jerking the head, while twisting it strangely to one side. In drinking, the head is plunged much too deeply into the bucket, often over the nostrils, and the water is actually chewed. When walking, the legs are frequently lifted very high, as though some obstacle were to be stepped over, and regular circular movements may be observed. In pushing backwards, the horse drags its fore legs after it instead of raising them.

3. *Disturbed Volition.* This is shown in various ways. The sick horse only obeys very slowly an order to move to the right or left in its stall, or not at all, is very dull and lazy at its work, must constantly be urged on and can only with great difficulty be got to move backwards, and often not at all. Instead of doing so, it rears up, will obey neither the rein nor the whip, but attempts constantly to move to one side.

4. *Disturbances of Sensation,* chiefly shown in diminution and dulness of the same. One may step upon the animal's coronet without it taking any notice, or may pull its ears, pinch its flanks, or clap the hands before its eyes, with hardly any result.

5. *Secondary Disturbances of the Heart, of Digestion and Respiration.* The pulse is soft, slow and often below its normal number, peristalsis is frequently interfered with and suppressed, as is also evacuation ; breathing is deep and slow and its frequency much below the normal rate.

**Ophthalmoscopic Result.**—As a constant and invariable accompaniment of sleepy staggers Lustig and Esberg mention hyperæmia of the optic papilla and of the adjacent vessels of the retina (choked papilla). We have ourselves only once observed such a hyperæmia by ophthal-

moscopic examination ; but, on the contrary, frequently found marked anaemia of the papilla, as also have Eversbusch and Berlin. We have usually found the papilla to be quite normal in sleepy staggers. Heyne reports the same result, having only twice demonstrated hyperæmia of the papilla in forty-three cases.

**Course.**—This is chronic, the condition often lasting for years and even until the animal's death. In other cases, distinct improvement or retrogression has been observed. The latter occurs mostly when unfavourable circumstances have reacted on the animal (over-exertion, too rich food, steamy stables, great heat). Thus in summer such horses often seem much worse, as compared with other seasons. On the other hand, favourable influences may cause marked diminution in the symptoms, which is especially the case after using pilocarpine, and thus make one believe that a complete cure has been effected ; but we must join with Gerlach in denying that this is possible.

Sometimes chronic hydrocephalus is complicated with congestion and even inflammation of the brain, especially when the chronic has been developed from an acute, inflammatory form. We then obtain the aspect of staggers plus that of brain-congestion or inflammation—which latter predominates. These fits of excitement, mania and raving are sometimes evanescent (congestion of the brain), sometimes more prolonged (inflammation of the brain). Formerly this complication was called "raving" or "erethistic" staggers, a name which should no longer be retained, and which one must remember means nothing but an acute (hyperæmic or inflammatory) process upon a basis of chronic hydrocephalus. Further description is unnecessary, as it corresponds entirely with hyperæmia and inflammation of the brain. Finally, we must observe that, though chronic hydrocephalus is not itself fatal, yet that, when complicated with cerebral inflammation, it may frequently have such a termination.

**Chronic Drapery of the Ventricles in Other Animals.**—The symptoms are not very different from those of sleepy staggers in horses. Cattle, pigs, dogs and sheep show persistent stupidity, heaviness in the head, and seem almost imbecile ; thrust to one side, grope their way uncertainly, run up against objects (amaurosis), are deaf and deficient in power of smell ; digestion also is disturbed, but there is no rise of temperature. The sickness is generally called "stupidity," and in pigs sometimes "silliness." It is not unfrequently a sequel of distemper in dogs, and is occasionally congenital. The Berlin dog hospital reported 29 cases out of 70,000 dogs treated in nine years.

**Therapeutics.**—No treatment with a view to cure is of any avail. The remedies formerly used and praised, including perforation of the olfactory bulbs in order to empty the brain-ventricles (Heyne), have all been proved useless. Pilocarpine and arecoline are also of no avail when the condition (hydrocephalus) is once fully developed, as there are no means of compensating the atrophy of the brain caused by pressure. The utmost they can do is to produce a temporary psychic improvement and thus render the animal a little more serviceable. But by prudent prophylactic measures one may prevent the disease from growing worse, and especially ward off its dangerous complications with hyperæmia and inflammation of the brain. Along with cool and airy quarters, one must particularly see that the animals are not over-worked and not too richly fed. They should have laxative foods (bran, green food, turnips) and even gentle laxative medicines, the latter especially in summer; for instance, a little neutral salt occasionally.

**Sleepy Staggers as a Warranty-Defect.**—Sleepy Staggers is more important in its character as a defect against which warranty can be demanded than in its clinical aspect. Although this forensic question scarcely belongs to a work on the medicinal treatment of animals, yet the chief points observed in judging sleepy staggers must here be summarised. The disease is forensically defined as a non-febrile, chronic, incurable brain-disease of horses, which manifests itself in disturbance of consciousness, of perception, of volition and of sensation, and which may vary greatly in its degree and complications. Its cause is usually chronic dropy of the ventricles of the brain. In rare cases the following pathological processes in the brain can also produce the morbid aspect of sleepy staggers: hydrocephalus chronicus externus, pachy-meningitis and lepto-meningitis chronica, adhesion of the cerebral membranes to each other, to the brain or to the roof of the skull, exostosis of the cranium, chronic encephalitis with softening or sclerosis, ependymitis sclerosa, new growths in the brain or venous plexus, e.g., cholesteatomata (but which often show no symptoms), peammonata, melanomata, oedema and parasites in the brain (hydatids). Diagnosis is easy when the symptoms are pronounced, but difficult when otherwise; but some of these difficult cases may be judged according to their subjective conception. The animals (having no fever) are examined first at rest, then during and after exercise (riding, driving). One has to consider, not only solitary phenomena, but all in their relation to one another, and special weight must be laid upon diminution of usefulness. This always exists in horses subject to sleepy staggers, even though for some kinds of work they may be used for a long time; but for general traffic they are always dangerous, because unreliable and subject to inflammation of the brain. By way of differential diagnosis the following diseases must be distinguished from sleepy staggers:—1. Congestion and inflammation of

the brain, which are characterised by injection of the mucous membranes of the head, high temperature of the skull and symptoms of excitement. In particular, acute hydrocephalus is distinguished by its acute and often feverish course, its combination of depression and exaltation, of anaesthesia and hyperesthesia, by loss of appetite, variability of the pulse, and distinct post-mortem lesions (hyperæmia and inflammation). 2. Severe and general fevers, especially infectious diseases (influenza, murrain, glanders). 3. Affections of the brain accompanying the change of teeth (gumæ reddened, mucous membranes of the head injected, youth) and change of coat. 4. Sexual excitement, so-called "seminal" and "maternal staggers" (periodical and connected with the sexual life). 5. Gastro-intestinal catarrh, liver-complaints, etc., so-called "stomach" and "liver-staggers." (Compare Schweinberger Sickness. Vol. II., p. 244.) 6. Restiveness (conscious obstinacy). 7. Phlegma, weariness, senile weakness, deafness, blindness. 8. Inflammation of the sphenoidal cavity and of the turbinate process of the ethmoid bone (Lustig).

The period of warranty is in Saxony 15 days, in Bavaria, Württemberg and Baden 21 days, in Hesse and Prussia 28 days, and in Austria 30 days.

#### HYPERÆMIA AND ANÆMIA OF THE BRAIN.

**Etiology.**—Hyperæmia of the brain is conveniently divided into congestive or active hyperæmia, with a rapidly occurring fluxional excess of blood in the brain; and an engorged or passive hyperæmia, with accumulation of blood in the brain owing to defective or impeded outflow of the blood.

**I. Congestive or Active Hyperæmia of the Brain** occurs especially in young animals, and most frequently in horses, dogs and sheep, many animals seeming to possess an individual predisposition. Its causes are: severe bodily exertion, driving or riding too fast, over-strain during breaking-in; extreme psychic excitement, e.g., during the sexual period, during a railway journey (so-called railway sickness), or after a change of quarters; abnormal activity of heart following hypertrophy; shock or injury to the brain and its membranes; prolonged action of strong sunlight upon the head (sun-stroke insolation), excessive summer-heat, hot, stuffy stables, severe chills, sudden changes of weather; the process of dentition, especially in dogs and horses (but to which, as a rule, too much importance is attached); collateral congestion of the carotid arteries, as a result of the exclusion of important arteries of the general circulation, as in thrombosis of the arteries of the thigh and pelvis (intermittent lameness), or in compression of

larger arterial capillary areas by abnormal gas-pressure in the stomach and bowels (acute flatulence of ruminants, wind-colic of horses); excessive eating after previous fasting or with but little work, indigestible food (leguminous, summer-wheat, rye, clover).

As a secondary symptom congestion of the brain occurs in the course of many diseases, such as in brain and infectious diseases (oftenest, perhaps, in the distemper of dogs) and in poisoning by narcotic and narcotico-acrid substances. Finally, previous brain-affections (inflammation, chronic dropsy of the ventricles) seem to leave behind a predisposition to hyperæmia of the brain.

2. The causes of **engorged or passive hyperæmia** consist in local compression of the jugular veins by tight collars, throat and head-straps, by securing the head too firmly when breaking-in, by too short bearing-reins, or by a very large struma (goitre), especially in dogs. Moreover, non-compensated valvular defects of the heart, as well as destructive changes in the lung (emphysema, induration) and compression of the lungs by accumulated fluids in the pleural sac all tend to impede the outflow of blood from the veins of the brain. In cases of abnormal gas-accumulations in the stomach and bowels a passive hyperæmia of the brain is set up along with the congestive form.

3. The causes of **anæmia of the brain** (by which we must always understand an arterial anæmia) are weakness of the heart; compression of the carotids by tumours or after ligation; embolic or thrombotic stoppage of the brain-vessels; diminution of the total amount of blood in the body, or of its most important constituents (anæmia, severe haemorrhage, chlorosis, leucæmia); a too rapid flow of blood from the brain to other parts, e.g., upon suddenly emptying a pleuritic exudation or gas-accumulation from the stomach or bowels; an increase of inter-cranial pressure caused by compression of the brain by accumulations of fluid, tumours or effusion of blood. A reflex spasm of the vessels of the brain after the action of external irritants can also, perhaps, as in shock, be regarded as a cause of anæmia of the brain. Finally, it forms an accompaniment of many diseases associated with poverty of blood. As passive hyperæmia of the brain always brings with it an arterial anæmia, it may also to some extent be regarded as a cause of the latter.

**Post-mortem Lesions.**—1. *Hyperæmia of the brain* is marked by great fulness of the brain-vessels and of its membranes, along with which small haemorrhages may also be present in the soft cerebral substance itself. The grey matter of the brain is a bluish-grey, and the white is dull grey to greyish-yellow. The cut surface of the brain shows a large number of blood-dots, which may be ranged in rows and, in contrast to the haemorrhagic foci in apoplexy of the brain, can be easily wiped off. The entire brain appears larger, in consequence of which, when the hard cortex is cut, it bulges somewhat. In severer stages of brain-hyperæmia, transudation of serum into the peri-vascular lymph-cavities takes place, and even into the cerebral tissue itself, whereby the capillaries of the brain are compromised. Viewed as a whole, this condition of cerebral oedema is characterised by poverty of blood in the whole organ, a singularly watery lustre upon the cut surfaces, and even the oozing therefrom of serum, and also by the diminished consistence of the brain substance. After frequently repeated hyperæmia, opacities and thickerings of the soft substance often remain, with enlargement of the vessels and thickening of the ependyma. Possibly the so-called Pacchionian granulations upon the arachnoidea are produced by chronic hyperæmia of the brain.

2. *Anæmia of the brain* consists in a notable emptiness of the blood-vessels of the brain and of its membranes, as a result of which its grey matter appears remarkably clear and colourless, and its cut surface shows only very few blood-dots. In cases of chronic anæmia the substance of the brain itself has often a somewhat tough consistence.

**Symptoms.**—In the active form of hyperæmia of the brain these are shown in paroxysmal appearances of excitement, in which disturbances now of the psychic and now of the sensory or motor spheres predominate, usually to be soon followed by signs of depression. The several aspects vary greatly according to the intensity and locality of the hyperæmia, or the individuality and species of the animal. The excitement stage often commences suddenly, but in some cases gradually, with signs of restlessness and agitation, which may grow to fury and mania. Horses thrust forward against barriers and walls, striking them with their heads or breasts and frequently sustaining serious injury; or they rear up, strike out with the fore feet on to the halter-chain or manger, kick

out without cause, bare their teeth, bite neighbouring objects, neigh and shake their heads. Others leap back suddenly in the stall, snap their chains and then generally tumble over backwards, or hang themselves in the halter. They are at the same time very timid, and extremely sensitive to external influences (touch, noise, light); not seldom muscular tremblings and even epileptiform spasms may be observed. Cattle step suddenly back from their stall, leap wildly to and fro, bellow loudly, butt with their horns, and often fall down with epileptiform spasms and twitchings, grinding of teeth, foaming at the mouth, and twistings of head, neck, eyes, etc. Dogs are extraordinarily restless and excited, howl and whimper, are given to biting, snap at flies, try to break loose, manifest uncontrollable movements, vomiting, twitchings and eclamptic spasms. Along with these psychic, sensory and motor evidences of excitement, the mucous membranes of the head are much redder, the pupils contracted, vision is disturbed, the eye often shining and fiery, and the ophthalmoscope reveals considerable active hyperæmia of the optic papilla; the temperature of the skull is raised, the pulse is full, its frequency increased and breathing quickened. In the stage of depression, which often soon succeeds, even in a quarter of an hour, the morbid aspect is completely reversed and has changed to a condition of stupefaction and dulness. The animal appears sleepy and inanimate, hangs its head, rests it on the bar or trough, has a staring, expressionless eye, takes no notice of its surroundings, strikes unnatural attitudes, and sometimes performs automatic movements (running round as in a circus, or pointing as though at game), or staggers and tumbles in walking. Appetite is diminished, intermittent or unnatural, urination and evacuation are delayed; the pulse is at times below, and at others above, the normal; the pupil is dilated. The course of acute hyperæmia of the brain is very varied. Sometimes complete recovery takes place in a few minutes or, after repetition of the attacks, in a few hours or days. At other times an inflammation of the brain develops from the hyperæmia, or rather the latter forms the initial stage of the former; or it may end in fatal apoplexy due to vascular rupture or œdema of the brain. Lastly, abnormal conditions, such as sleepy staggers or vertigo, may remain permanently. The prognosis must therefore always be given with caution.

2. The symptoms of **passive hyperæmia of the brain** agree in the main with those of the second stage of the active

form, and are chiefly those of depression. But they are not rarely interrupted by signs of excitement. The morbid aspect is also frequently that of cerebral anaemia.

3. In acute cases of anaemia of the brain the symptoms consist in the occurrence of vertigo, which may increase even to insensibility (syncope) and apparent death. We notice weakness of heart, small pulse, vomiting (in carnivora and omnivora), dilated pupils, paleness of the optic papilla (revealed by the ophthalmoscope). Sometimes the apparent death becomes real: so-called nervous stroke (apoplexia nervosa). But under some circumstances the aspect of anaemia of the brain may be altogether identical with that of acute hyperæmia, and present great excitement followed by stupor; but this happens more frequently in cases of chronic anaemia of the brain.

**Differential Diagnosis.**—It is difficult to distinguish hyperæmia from inflammation of the brain, the symptoms of both being exactly alike, and, as a matter of fact, in not a few cases differentiation is quite impossible. In general, a slighter degree of intensity and a more favourable course, coupled with the absence (which is not invariable) of a feverish rise of temperature, point to such cases being hyperæmia rather than inflammation.

**Therapeutics.**—In addition to the removal of all causes of the disease, the treatment of *acute hyperæmia of the brain* demands an energetic attempt to prevent the excessive flow of blood to the brain. In this respect liberal blood-letting is the best remedy, if it can be done promptly at the outbreak of the disease; but later, when signs of depression have already appeared, it must be condemned. Besides this, the application of cold to the head is to be commended, and can best be done by an ice-bag, but must be occasionally suspended. If no ice be at hand, pour cold water on the skull. Finally, we attempt to divert the blood from the brain to the intestines by derivatives. But drastic remedies must only be used at the very beginning of the attack, and for young and well-nourished animals. If the symptoms of excitement have passed away with the administration of drastics, then, according to our experience, the depressional stage will only be prolonged in consequence of too great a diversion of blood from the brain to the bowels, and the whole condition be thereby rendered worse. We therefore usually only give the milder laxatives, especially

neutral salts and small doses of tartar emetic. Their operation may be assisted by cold clysters (infusions). Any derivation to the skin by irritant inunctions is rarely possible, owing to the timidity and excitability of the animal. Rather must all external irritants be withheld, and a quiet, dark, cool and airy room be provided. Give horses, if possible, a loose-box. Finally, see that cooling and laxative food is supplied : for horses the best is green fodder, turnips and bran.

2. The treatment of *anaemia of the brain* is the contrary of that just specified. Especially during unconsciousness, which is its most frequent manifestation, the application of stimulants is imperative, viz., ether, alcohol, spirits of ammonia, camphor, coffee, caffein, hyosciamine, atropine, strong skin-irritants, etc. In chronic anaemia of the brain, the general anaemia must be treated in accordance with its causes.

#### CEREBRAL HÆMORRHAGE. APOPLEXY OF THE BRAIN.

**Miology.**—Apoplexy of the brain, also known as blood-stroke, stroke, brain-stroke, seizure, etc., consists in rupture of one of the larger or smaller vessels of the brain, with effusion of blood and compression or destruction of the brain substance. It is produced by various causes. First a cerebral haemorrhage may occur in connection with hyperæmia or inflammation of the brain, so that the causes of the latter would have to be considered. In other cases vascular lesions form the starting-point of an apoplexy of the brain, e.g., atheromatous and fatty degeneration of the cerebral arteries. These are, however, less frequently the causes in themselves of ruptures, than in conjunction with an increased blood-pressure. Traumatic influences, and especially shock to the brain, chiefly cause haemorrhage in the cerebral membranes, particularly between the dura mater and the cranium (haematoma of the dura). Not unfrequently after emboli, hemorrhagic stoppages occur in the brain, just as in other organs. Thus we observed one in a dog in connection with metastatic formation of carcinoma of the lungs. Finally, haemorrhage on the brain takes place in general disorders of hemorrhagic character, such as anthrax, petechial fever, distemper, the typhoid of birds, poisoning, etc.; also in rupture of the heart and intrusion of parasites into the brain (cercaria).

**Post-mortem Lesions.**—Cerebral haemorrhage is com-

most in sheep, cattle and dogs, and rarer in horses. Its favourite localities in the brain are the vicinity of the lateral ventricles, the grey matter in the cerebral cortex, as well as the corpus striatum and the optic thalamus. Sometimes we find very small, so-called capillary, dotted haemorrhages, and at others larger or apoplectic foci.

The *capillary haemorrhages* are small, round or elongated blood-foci of a dark-red colour, varying in size from a millet-seed to a pea. The cut surface of the brain is sprinkled or streaked by them, and these dots cannot be wiped or washed off, differing in this from those which appear during a simple hyperæmia. If the discharged blood be still within the sheath of the pia of the vessels, these capillary haemorrhages are termed miliary dissecting aneurysms.

The *apoplectic foci* attain to the size of a pea or hazel-nut even on the rupture of the smallest artery, but may reach very large dimensions according to the size of the vessel implicated, so that extensive areas of the brain-substance are often destroyed by pressure. The recently formed foci present a dark, black-red, coagulated mass, which is soft or pulpy, and the surrounding brain-matter is often also softened. In older foci the blood-coagulum is contracted and lighter in colour, and the adjoining tissue has been stained yellow by diffusion of the colouring matter of the blood. Later on, the extravasated blood and injured brain-matter are gradually re-absorbed, after previous decomposition, and either a smooth-walled, free space is left, which becomes filled with fluid (an apoplectic cyst), or the brain-substance undergoes shrinkage by formation of additional connective tissue (an apoplectic cicatrix), which is stained yellowish-brown by pigmentation from the colouring matter of the blood.

**Symptoms.**—The signs of cerebral haemorrhage occur suddenly. They consist in stupor of the sensorium, vertigo, staggering and stumbling, trembling, involuntary movements (pressure to one side, running round in a circle, pointing, as at game), unconsciousness, collapse, raging. Less frequently the psychic powers remain undisturbed. The mucous membranes of the head appear very red; in fact the haemorrhage may extend to its tissues and outer surface, so that bleeding occurs from mouth and nose. The pulse is very weak to the point of imperceptibility, breathing dyspnoeic; and sometimes involuntary urination and evacuation may be noted.

These signs may be complicated from the first or later on with paralytic symptoms, which, by their peculiarity, point to some local affection of the brain; so-called focal symptoms. Among these are paralysis of single muscles or groups of muscles (monoplegia), e.g., one-sided paralysis of the muscles of lips, tongue, jaws, eyelids or ears, disablement of a hind or fore leg; further, semi-lateral paralysis (of the half of one side, or hemiplegia), paralysis of the optic nerve with subsequent amaurosis, paralysis of sensation throughout an entire half of the body (hemi-anesthesia), paralysed deglutition (haemorrhage in the medulla oblongata). Monoplegia indicates an affection of the motor centres of the cerebral cortex; hemiplegia points to haemorrhage in the region of the path of conduction up to the pyramid, both upon opposite halves of the brain. But only too frequently a certain localisation of the focal symptoms is impossible. The course of apoplexy of the brain may be fatal in a few minutes or hours, but days and weeks may pass before the end, during which the attacks are sometimes repeated. Cure is rare and usually incomplete, paralysis remaining behind.

**Diagnosis.**—The recognition of cerebral haemorrhage is based upon the sudden occurrence of severe cerebral attacks, complicated with signs of congestion of the brain and paralysis. In contrast to these, in so-called lung-stroke (extreme, active hyperæmia of the lungs with oedema), the symptoms of disturbed respiration are most prominent. The apoplectic form of anthrax, which presents the same phenomena as apoplexy of the brain, may be distinguished by microscopical examination of the blood. Finally, a brain-stroke may be confused with several forms of poisoning (prussic acid, strychnine). In such cases autopsy and chemical tests must decide.

**Therapeutics.**—The treatment of cerebral haemorrhage is somewhat hopeless. Phlebotomy can only be justified during the stage of hyperæmia of the brain; later it must be rejected as harmful. The application of cold after the congestion of the brain has diminished is hardly of much use. But, on the other hand, something may be attempted, as in brain-anæmia, with stimulating remedies, so as to relieve the depression of brain activity; for which give ether, alcohol and camphor. When the cerebral hyperæmia is of long duration, derivation to the bowels may be so far advisable, as that it

prevents haemorrhage. The surviving paralyses are to be treated with massage, electricity and with strychnine. Iodide of potassium may be administered internally, in order at least to try and hasten resorption of the extravasated blood.

#### STURDY OR STAGGERS (GID OR TURNSICK).

**Occurrence.**—Among parasitic diseases of the brain staggers is the most important. It is commonest among sheep and, in the second place, in cattle; only rarely is it seen in horses, goats, antelopes and also in rabbits and hares.

**Natural History Notes.**—The cause of staggers must be sought in the presence within the brain of *cœnurus cerebralis*, the bladder-worm of the brain (staggers-worm, or brain-bladder); in rarer cases these latter are also found in the spinal medulla. As Küchenmeister first proved by experiment in 1853, *cœnurus cerebralis* represents the "nurse" of *tænia cœnurus*, a tape-worm which especially infests sheep-dogs and butchers' dogs. The sheep are infected by the proglottides or eggs contained in the dung of such dogs, which, dropped in the pastures, are picked up by them. As these eggs retain their vitality longer in damp seasons, the disease is more prevalent in such years. Sheep-dogs, on the other hand, are infected by eating the brains of sheep suffering from staggers. The development of the *cœnurus*-cysts occurs by liberation of the six-hooked embryo through dissolution of the tape-worm egg-shells in the sheep's stomach. The embryo then penetrates the membranes of stomach and bowels and wanders towards the brain, or spinal marrow; whether along the circulatory channels or by following the connective tissue is not yet decided. According to Müller, it is most likely that the brood reaches the brain by embolic methods through the blood. The certain proof of verminous emboli in the brain, and the fact that inflammatory changes are never found upon the basilar surface of the brain, seem to support this theory, especially as such changes are common upon the upper surface of the hemisphères, and the dura is never found to be perforated. When once the embryos have settled in any part of brain or medulla, they lose their hooks and gradually change into bladders or cysts. After 14 to 19 days they are the size of a millet or hemp-seed; in 26 to 42 days they reach that of a pea and in

50 are as large as hazel-nuts. They attain their organic development in 2 to 3 months.

**Post-mortem Lesions.**—1. During the *emigration-stage* of the embryos into the brain, which represents clinically that of brain-irritation, we find on the upper surface of the cerebral hemispheres, in the reticulate tissue of the pia, round stripes, intensely yellow, about  $\frac{1}{3}$  inch thick, which run sinuously upon the diagonal section. These, according to Möller, consist of a central haemorrhage with a peripheral accumulation of pus-corpuscles, and therefore represent a localised haemorrhagic purulent lepto-meningitis. Upon the corresponding place on the dura a similar fibrino-purulent pachy-meningitis is also present. These serpentine lines are nothing else than the immigration routes of the coenurae, at the terminus of which they may be found as cysts, from the size of a pin-head to that of a pea, usually several together (up to ten, Huzard says as many as thirty). Similar pathways are found in the brain itself in the form of winding passages, full of purulent, creamy fluid (*encephalitis suppurativa*). The embryos may even reach the choroid plesus, when suppurative hydrocephalus internus is set up, with swelling and suppurative infiltration of the plexus, as well as purulent proliferation on the part of the ependyma. Besides these, necrotic foci,  $\frac{1}{2}$  to  $\frac{1}{4}$  inch wide, occur in the brain, probably caused by verminous emboli. Finally, we find both in brain and plexus haemorrhages, both small and large, and even extensive apoplectic foci.

2. In their *fully developed stage*, which is that of true staggers, we find the coenurus-cysts grown to the size of pigeons' or hens' eggs. Their shape varies from round to oval (in the spinal medulla a longer oval or even cylindrical), and they contain a thin fluid as clear as water. Their inner walls are covered with numerous vesicles (up to 500), which may make them bulge outwards. There is either one such large cyst or bladder, or there may be several (4 to 6) smaller ones, which displace the brain-substance and may even erode the bone of the cranium to the thinness of paper, so that it may be perforated. The brain immediately adjacent is anaemic and sclerotic (atrophy of the brain). Very large cysts may occupy the entire half of a brain (Reboul). In addition to these changes in the brain, we may not unfrequently discover dead and calcified coenurae in the connective tissues of the body, in the subcutis, muscles, heart, liver, lungs, kidneys, mesentery, etc. These are granular and fatty-de-

enerated nodules, from a millet-seed to a pea in size. Fully developed coenuræ have also been found under the skin.

**Symptoms of Staggers in Sheep.**—This disease, which is known by many local names, occurs in hardly any but young sheep (lambs and yearlings), and is exceptional after their second year. In wet seasons especially it is almost epidemic and plays great havoc in a flock. Even new-born animals may have staggers, caused by embryos received from the mother during pregnancy. Three stages of the disease must be distinguished.

1. The stage of brain-irritation, following on the immigration of the embryos, the so-called "Staggering." Time: late summer to autumn; duration: 8 to 10 days. 2. The stage of incubation, lasting from 3 to 6 months. 3. The stage of the true staggering sickness, commencing in winter and on towards spring, and lasting from 4 to 6 weeks.

1. The stage of brain-irritation is only observed, according to Möller, in about one-fifth of the animals which later have staggers, and its symptoms vary according to the extent of the immigration. Very often only trifling disturbances occur, such as melancholy and lassitude, a clumsy gait, stupid, expressionless look, or lagging behind the flock. In severer cases the brain-phenomena are much more intense. The mucous membranes of the eyes, especially the conjunctiva sclerae, are very red, the top of the skull feels hot, the animals hang their heads, are noticeable for great timidity and excitability, rush off suddenly or run aimlessly hither and thither, press to one side, run in a circle or turn round and round, are dizzy, stagger and easily tumble, and fall into spasms and fits of twitching. This condition may end fatally in 4 to 6 days with increase of stupor (according to Möller in 5 per cent. of all cases). But with most, the symptoms abate after 8 to 10 days, and in a few cases (not more than 2 per cent., says Zürn) complete recovery takes place. Generally, though, there is merely a prolonged pause in the disease.

2. The second stage of incubation usually passes without any noticeable symptoms. The animals are apparently healthy; but on closer observation one may sometimes perceive a certain diminution in their intelligence.

3. The stage of true staggers commences with gradually increasing heaviness and stupor. The animal suddenly stands still, ceases unexpectedly to eat, or stops eating altogether, is dull and

faint, runs unconsciously against things, has a glassy, staring eye, with dilated pupil, and manifests peculiar characteristic motor-disturbances. The several unconscious and uncontrollable movements are as follows : (a) Running as in a circus, making smaller or larger circles round a central point in one or other direction ; (b) twisting or pointing movement, in which only the fore or hind quarters are turned round a hind or fore leg, the head and neck being sunk ; (c) the rarer rolling movement, in which the animal, lying on the ground, rolls round the long axis of its body ; (d) in which it sways to one side, reels and often falls down—so-called “dizzy or tumbling staggers” ; (e) in which, with lowered head and legs uplifted, it runs quickly straight before it—so-called “trotting staggers” ; and (f) when it runs rapidly straight on, with the head raised or even bent to one side, frequently stumbling and even falling over backwards—so-called “sailing staggers.” Of great diagnostic importance is the formation of a circumscribed area on the cranium, which is more yielding than the rest and even fluctuating. Pressure upon this generally causes pain and even convulsions. Unless the animals are early killed or operated upon, death takes place in four to six weeks from paralysis of the brain, accompanied by growing emaciation and feebleness.

**Rump-staggers.**—The immigration of coenurus-cysts into the spinal medulla produces the symptoms of so-called “rump-staggers.” These consist at first in weakness of the loins, swaying to and fro of the hind-quarters when walking ; a gait resembling that of fowls, with the legs thrust under the body ; pain and even collapse on pressing the flanks ; and finally, complete paralysis of one or both hind legs, which are dragged along as though dead. The animal perishes at last from general wasting and debility. Loss of consciousness, or any involuntary motions, are only noticed when there is also a cyst in the brain.

**Symptoms of Staggers in Cattle.**—Contrary to our experience with sheep, this malady is not rare in older animals among cattle, though still most common in yearlings. It begins with irregularity in the taking of food, the animal suddenly breaking off while feeding. Then timidity is shown, laziness in movement, together with a peculiar posture of the head, it being lifted up and held sideways with a quick succession of twitching movements. The glance is staring and stupid, and the pupils are dilated. The root of the horns and the forehead feel hot ; breathing and pulse are accelerated. To these come disturbances of movement : the beasts press forward, and it is difficult or impossible to make them step back, or they show decided

"circus" or turning movements. Sometimes they tumble down suddenly in their stalls, when they may even wrench off their horns, and show maniacal signs and spasms. Percussion of the skull should be carried out, after previous removal of hair from the forehead, with the metal back of the percussion-hammer and very carefully. Sometimes it detects a somewhat duller tone on the site of the cyst, and reveals great painfulness of the surrounding part. The result of the disease is the same as in sheep, but the course much more rapid. Compulsory slaughter is generally the rule, and, according to Rudolfsky, this is oftenest necessary in the months of May and June. From the first symptoms to the climax of the disease four to five months may elapse.

**Staggers in Horses.**—This is exceedingly rare, and occurs at first with the aspect of sleepy staggers or inflammation of the brain. These signs are complicated with rotary movements around one leg, running in a circle, backing, dizziness, blindness, etc. Death sometimes occurs apoplectically.

**Differential Diagnosis.**—Staggers may be confounded with a number of other complaints and, especially at its outbreak, with cerebral inflammations arising from other causes, such as that produced by the larvæ of oestrus (*oestrus ovis* sometimes attains the brain); also with epilepsy, vertigo, blindness, suppurative catarrh of the antrum; and in addition to these cerebral tuberculosis in cattle and hydatid disease (*echinococci*). It is naturally impossible to distinguish it from these two last diseases during life; but it may be known from that caused by *oestrus-larvæ*, which is sufficiently characterised by the catarrhal processes on the part of the mucous membranes of the nose and frontal cavity. We may also exclude inflammation of the brain, in so far that it does not generally occur in the form of a focal disease. When once staggers has been shown to be present in a herd, the diagnosis of further cases is not difficult, and one will not go far wrong in taking every case of brain-disease in the same herd to be one of staggers. Confusion of rump-staggers with trotting-sickness need not occur, if we take into account the other distinguishing signs of the latter, and especially the so-called nibbling.

**Prognosis.**—The apparently favourable signs during the second stage of staggers are really a very unfavourable indication, because the disease, if uncombatted, is sure to end fatally,

and because all remedies we can apply are only able to save about one-third of those attacked. For this reason sheep which are bred for fattening had better be slaughtered at once. Opinions vary much as to the value of the treatment, which is purely operative; and we share the opinion of Dammann, Möller and others, that it can never prevent very heavy losses, and ought not therefore to be thrust into the foreground whenever the disease appears in epidemic form.

**Therapeutics.**—Apart from prophylaxis, the treatment of staggers is essentially surgical and consists in removal of the coenurus-cysts from the brain. This very old operation, known among shepherds as "staggers-boring," is more suited for sporadic than for enzoötic outbreaks. The reason why this plan only succeeds in saving about one-third of the affected animals lies in the fact that the cyst often cannot be found, or lies too deep for removal, and that in quite a third of the cases there are, not one, but several cysts. Moreover, haemorrhage or suppurative inflammation may follow the operation, and, finally, in many cases the anatomical and functional disturbances of the brain have gone so far that nothing can save them. For a successful performance of the operation two factors are important: the localisation of the cyst and a suitable method of operation.

**I. General Diagnostic Indications for the Situation of the Cyst.**—If this cannot be fixed either by palpation or percussion, one may be guided by the nature of the motor-disturbances and by the position in which the head is held. Thus when it is held sideways, that part which is lowest usually corresponds with the site of the cyst. In the course of time, and as a result of many comparisons of the clinical phenomena, certain diagnostic indications have been gained, but which, we must premise, often lead us astray. The diagnosis in this respect can, therefore, only be one of probabilities. The several indications are as follows:

- (a) In what we have called "*circus-movements*" the cyst lies superficially in the large cerebral hemispheres, and upon the side nearest the centre of the circle described by the animal.
- (b) In right-handed "*pointer*" movements, either upon or far within the right hemisphere, or else upon the floor of the left ventricle; when left-handed then upon the reverse side.

- (c) In "*trotting*" staggers at the anterior end of the hemisphere (frontal lobe).
- (d) In "*dizzy*" staggers laterally in the lobe of the cerebellum or cerebrum.
- (e) In "*sailing*" staggers between the cerebrum and cerebellum.
- (f) In "*rolling*" staggers at the base of the cerebellum.

2. Of **methods of operation** only two can be considered, viz., trephining and use of the trocar. (a) For *trephining* sheep we use a crown about  $\frac{1}{2}$  inch in diameter, and for cattle the usual instrument for horses; the central line of the skull must if possible be avoided, owing to the longitudinal sinus beneath it. When the position of the cyst cannot be externally fixed, the chief spots for operation are: in male sheep,  $\frac{1}{2}$  inch behind the middle of the horn; in females,  $\frac{1}{2}$  to  $\frac{3}{4}$  inch behind the process of the horn, and the distance from the median line should be at least  $\frac{1}{2}$  inch; this spot corresponds with the centre of the posterior lobe. The spot immediately behind the inner corner of the horn or of its process (hinder part of the anterior lobe) is only to be considered as of secondary importance. For the operation itself on sheep and cattle, see the text-books of surgical instruction.

(b) The *trocar* should be used upon sheep so that the cyst may be pierced and emptied by suction, and then the wall of the cyst may also be sucked out and removed. According to Zeden, the trocar is first inserted a finger-breadth behind the horns and the stiletto withdrawn, when a portion of the watery contents flows out of itself, the rest being extracted by a suction syringe inserted through the canula. The wall of the cyst is then drawn out by the syringe and pulled away with forceps. Dammann has modified this method, in that he only inserts the trocar at first  $\frac{1}{2}$  inch deep, and only pierces further when he fails in this way to reach the cyst; moreover, the point of the trocar should be directed somewhat inwards. In case of need all the four spots named above and even the centre may be tried. Erdt's modification consists in using a punch for thicker skulls and introducing the trocar obliquely from the front inwards, a finger's breadth from the horn backwards and an equal distance from the median line; the clefts of his canula are serrated for better laying hold of and extracting the membrane of the cyst.

3. **Prophylaxis.**—Obviously more important than any

surgical treatment is the prevention of the disease. This lies in destroying as far as possible the tape-worms of the sheep-dogs, which should be attended to several times a year; also in burning or boiling the brains of all sheep that suffer from staggers. In districts where the malady is prevalent, it would be well not to let lambs or yearlings pasture, but to feed them in the stables, in spite of any extra cost.

**Rolling-sickness in Dogs.**—This may perhaps best be discussed in connection with staggers. It consists in fits of rolling which seize otherwise apparently healthy dogs, during which the head is at first held aslant and the whole body revolved several times around its longitudinal axis. There seem to be no disturbances of consciousness. The causes of this ailment, which evidently springs from an affection of the cerebellum, are various, but in general are little known. In a few cases we have found haemorrhagic foci in the temporal lobe and on the crus cerebelli, which were of traumatic origin. Not unfrequently this rolling-sickness has been observed during nervous distemper and inflammation of the brain as an expression of some focal affection in the region of the cerebellum. In a solitary case we noticed the condition as a reflex phenomenon caused by constipation (relapsing). Mauri blames an embolus in the vessels of the cerebellum, having in two cases demonstrated a circumscribed softening of the same. As regards treatment, we obtained relatively the best results by administering sulfonal (8 to 30 grains), hypnon (4 to 30 grains) and urethan ( $\frac{1}{2}$  to 5 drams). Among 70,000 dogs treated at the Berlin dog-hospital in nine years, only thirty-three suffered from rolling-sickness.

## TUMOURS IN THE BRAIN.

**Occurrence.**—These, which on the whole are rare, appear most frequently in the choroid plexus in the form of chole-steatoma or margaritomata (which often produce no disturbance), melanomata, psammomata, fibromata and cysts, also as oedema proliferans. Then also upon the cerebral membranes as sarkomata and lipomata of the dura, epitheliomata, dermoid and dental cysts; in the brain substance as gliomata, glio-sarcomata and odontomata; and finally, as exostoses, sarcomata and carcinomata of the cranial walls. With regard to tuberculous tumours, see the chapter on tuberculosis (Vol. I., pp. 152-203); respecting parasites, the paragraphs on inflammation of the brain, sleepy staggers and staggers (the earlier parts of the present chapter).

**Symptoms.**—These sometimes resemble those of general encephalitis, sometimes those of sleepy staggers or apoplexy of the brain. Focal symptoms especially are a characteristic

sign up to a certain degree, but yet are often absent. Just as has frequently been observed in injuries to the brain, so in this case also the several parts of the brain react quite differently to tumours. If these lie in the less important cerebral regions, such as the mesocephalon or corpus striatum, they may remain a long time without giving rise to any distinct symptoms. These are only developed when functionally important parts are attacked, such as the hemispheres of the cerebrum, with their psycho-motor centres, or the base of the brain with the places of exit of the twelve cerebral nerves. Such functional disturbances are usually restricted, owing to the limited nature of the tumour, and are known as focal symptoms. Without such focal symptoms, which gradually increase, and for which no internal or external cause can be traced, no opinion respecting the presence of a brain-tumour can well be formed during life, and a certain diagnosis is impossible. The following symptoms have been indicated in veterinary literature :—Twisting to one side, vertigo, disturbed power of balancing, epileptiform fits, psychic excitement, amaurosis, one-sided atrophy of the muscles of deglutition, hemiplegia, hemi-atrophy, hemi-anæsthesia, local muscular paralysis (monoplegia), etc. Of importance for diagnosis is the participation of the brain-nerves in the paralysis, viz., the nerves of the optic muscles, the visual nerves, the motor fibres of the trigeminus (masseteric nerves), of the *facialis* and *hypoglossus*. This indicates a basal position of the tumour. Respecting the occurrence in domestic animals of an engorged optic disc, such as is characteristic of brain-tumour in man, nothing is yet known. This is explained by the increased pressure within the brain caused by the tumour, whereby the cerebro-spinal fluid presses into the lymph-sheaths of the *opticus*, thus giving rise to an œdema of the lamina cribrosa, with secondary compression of the papillary vessels and venous congestion in the papilla.

#### SUNSTROKE AND HEAT-APOPLEXY.

**Definition and Nature.**—Whereas until recently these two names were considered as almost synonymous, later researches have shown that the two conditions must be differentiated, since they represent different affections. Although opinions are still divided as to the exact nature of both, yet the following may be laid down respecting their relation to each other.

1. **Sunstroke** (*the coup de soleil* of the French) is an affection of the brain caused by the direct action of the sun's rays upon the skull (brain and medulla oblongata). In slighter cases we have merely cerebral hyperæmia with its corresponding symptoms. But in severer ones we meet either a paralysis of the vital centres in the medulla oblongata, especially the respiratory, or else inflammation of the brain or a sanguineous apoplexy of the same.

2. **Heat-stroke or Heat-apoplexy** (*coup de chaleur* in French) is caused by over-heating of the body, combined with over-exertion and impeded outflow of bodily heat. It occurs mostly in horses which have severely exerted themselves during great heat, as in war or during manœuvres, also in pigs, sheep and cattle when driven in herds. Fat pigs, unaccustomed to transport, are specially susceptible. Many cases hitherto reckoned as sunstroke evidently belong to this category. Respecting the nature of heat-stroke and the cause of death there is as yet no unanimity of opinion. Some maintain that the chief cause is a change in the blood (destruction of the red blood-corpuscles, acid reaction of the blood, excess of carbonic acid, lactic acid or of urea, uræmic auto-intoxication). Others blame severe disturbance of innervation in the form of interrupted regulation of heat with abnormally high internal temperature and opaque swelling, especially of the cerebral ganglionic cells. (The temperature rises to  $109.5^{\circ}$  Fahr. and above.)

**Symptoms.**—1. In *sunstroke* these usually appear suddenly and without warning. In contrast to heat-stroke, there is no feverish rise of temperature at the beginning, nor often during the whole course. The symptoms differ according to the changes in the brain. Either we have the aspect of hyperæmia or inflammation of the brain: restlessness, excitement, mania, spasms, trismus, vomiting; or sudden death occurs like apoplexy without any further signs; or, lastly, the animals die with the phenomena of growing paralysis of respiration. In a case of sunstroke in a horse reported by Bartke, signs of restlessness, pawing, extreme redness of the mucous membranes of the head, violent mania and attempts to vomit were manifested; death occurring in three hours. Benjamin observed pronounced symptoms of rabies in a dog which had been exposed all day to a blasing sun. Siedamgrotzky saw a dog which lay chained up on a hot July day in the sun, and which suddenly died with-

out any symptom. The upper surface of its brain was strongly injected and infiltrated with small haemorrhages ; between the dura and arachnoid lay a considerable quantity of bloody-serous fluid ; and the cut surface of both brain and medulla oblongata was covered with numerous minute haemorrhages. Another important point in differentiating sunstroke from heat-stroke is, that the former occurs without any previous exertion or movement.

2. The symptoms of *heat-stroke* are : weariness during work, profuse sweating, great rise of internal temperature, extreme difficulty of breathing, much timidity, palpitation, rapid pulse, which grows weak or imperceptible, at first dilation and then contraction of the pupil, reeling, staggering, swooning, collapse, trembling, twitching, jerking of the legs, with gradual approach of death. Recovery is rare. On *post-mortem* the blood is found of blackish-red colour and thick consistence, the muscles are dry and hyperæmia is present in the left heart, and especially in the lungs. If animals are subjected to severe exertion in hot sunshine, the symptoms of both diseases may be combined.

Bartke has reported fully on several cases of heat-stroke in military horses, mostly occurring during the September manoeuvres in hot, close weather and after severe exertion. All the sick animals showed great weariness ; a few died before they could reach their stalls. Others staggered and fell in the stable, showing very hard breathing and profuse sweating. In all of them palpitation was marked, and in some was such as to shake the whole body and to be audible some distance away. The visible mucous membranes were dark red. Death was in many cases rapid and appeared to be due to asphyxia. The internal temperature in those which recovered rose to  $105^{\circ}$  F., and remained a full week at this point ; they were not fit for service until after five weeks.

**Differential Diagnosis.**—For the differentiation between sunstroke and heat-stroke the most important points have been already given. But both maladies may be confounded with hyperæmia, inflammation and apoplexy of the brain ; with vertigo, swooning, epilepsy, anthrax, some forms of erysipelas and of poisoning. In pigs the possibility of confusion with erysipelas is of special practical (forensic) importance, and a decision can only be secured by autopsy. In heat-stroke the changes shown are mostly negative, as compared with the very

characteristic marks of erysipelas :—gastro-enteritis, swollen liver and spleen, nephritis and bacilli in the blood.

**Therapeutics.**—The treatment in both diseases consists in reduction of the heat (see that for inflammation of the brain), also in quiet, supply of water, and administration of stimulants, especially the subcutaneous injection of camphor, ether, alcohol, atropine, hyoscamine, caffeine, veratrine, strychnine, etc. Chinin has also lately been recommended, and if the heart-beat be excessively rapid, chloroform.

## LIGHTNING-STROKE.

**Occurrence.**—Animals pasturing in the open, such as cattle, horses and sheep, are most liable to be struck by lightning ; also military horses. The effects vary with the intensity of the flash and its direct or indirect action upon the several animals.

**Symptoms.**—The operation of a lightning-stroke either shatters the nervous system electrically, or burns the skin, or rends the softer parts of the body. Strong shocks kill instantly, as though by apoplexy, through paralysis of the nervous system. Weaker ones, or those which strike near at hand, produce various conditions of stupefaction and paralysis. Thus a state of stupor, such as we notice in cases of concussion of the brain, may remain for several hours. In other cases paralysis of single extremities is left behind (of an ear, of one side of the lower lip, of the swallowing muscles, or of an entire hind limb) in the form of paresis and paralysis. The paresis is marked by a relatively more favourable course. Not unfrequently lightning causes external injuries. On the skin we may also observe striped or fork-shaped branching lines or figures, which indicate the path of the current, and are caused by dissolution of the haemoglobin of the blood-vessels and its diffusion through the adjacent parts. Or the stream scorches up the prominent protective hair over a wide area, such as the eyebrows, forelock, etc. Along with this superficial singeing are deeper-lying burns of the cutis, sub-cutis and muscles, with laceration and blackening of the latter.

**Post-mortem Lesions.**—Beyond local changes the condition of a body killed by lightning exhibits little that is characteristic. The chief thing is a great fulness of the venous

system, dark, thinly fluid blood, rapid decomposition of the carcase, incomplete rigor mortis and also small haemorrhages in the inner organs (brain, kidneys, lungs, etc.), and beneath the serous membranes. Occasionally nothing at all abnormal is met with.

**Therapeutics.**—These are solely symptomatic. Stupefaction requires stimulants (camphor, spirit, ether, ammonia, veratrine, atropine, caffeine, hyoscyamine); and for the paralysis we apply skin-irritants (friction, massage, electricity, with sharp and stimulating embrocations).

## CHAPTER XVI.

### II.—DISEASES OF THE SPINAL CORD AND OF ITS MEMBRANES.

#### *Cerebro-spinal Meningitis. Spasms of the Neck.*

**Etiology.**—Mid-way between diseases of the brain and of the spinal cord is spasm of the neck or cerebro-spinal meningitis, which might therefore just as well have been included among the former. As its name indicates, it is an inflammation of the membranes of the brain and spinal cord, though chiefly only of the anterior portion of the latter, and agrees perfectly, both in nature, symptoms and anatomical changes, with spasm of the neck in man. The causes in our domestic animals are evidently to be sought in infection, as in by far the larger number of cases the malady appears enzootically and even epizootically. For this reason it might very well be treated among infectious diseases. But we place it here, partly because little or nothing is known of the infective agent, and partly because it is so frequently mistaken for inflammation of the brain. The causes which were formerly generally blamed, especially in sporadic cases, such as chill, cold, bad, stormy, or raw weather, over-heated stables, too early loss of coat, or food that is too rich, are all only of secondary importance. As is the case in man, so also in domestic animals, cerebro-spinal meningitis presents a somewhat inconstant and changeable aspect when individual cases are compared, not only in the various genera of animals, but specially in one and the same species.

**Occurrence.**—Of our domestic animals horses and sheep are most frequently attacked by this disease, then cattle, goats and dogs. Among these it is especially younger animals that suffer, particularly lambs and yearlings, as well as young and vigorous horses; moreover, an enzootic outbreak not

seldom occurs in the spring. During such an epidemic both horses and cattle may suffer together. The disease is said to have been first observed in America during the early fifties of last century, and was described by Large in New York. The earliest German notices date from about 1865. In 1876 the disease spread as a plague all over Egypt, and, according to Apostolides, 5,000 horses, 700 mules and 200 asses died in Cairo and its neighbourhood alone. Saxony, Silesia, Hungary and Russia have more recently had similar visitations.

**Post-mortem Lesions.**—The chief anatomical changes are at first a serous, and later a suppurative lepto-meningitis of the brain and commencement of the spinal cord. Consequently we find between the dura and pia mater of the brain, medulla oblongata and spinal cord, as well as in the ventricles of the brain, either a yellowish, turbid, serous fluid or a greyish yellow, milky, purulent and even fibrinous exudation, and this more especially on the basilar surface of the brain. Roloff also found in sheep a suppurative infiltration of the blood-vessels of the pia, particularly in their adventitia. Occasionally the projecting nerve-roots are discovered completely enveloped in purulent exudation. The upper surface of both brain and spinal marrow is oedematous and softened, and the external layer of the brain infiltrated with pus. We find also isolated foci of softening in brain and spinal medulla, and not rarely smaller or more extensive haemorrhages in the membranes and within both brain and spinal cord. The vessels, especially those of the pia, brain and spinal medulla, are greatly congested. Along with these signs, though not in all cases, general bodily changes have also been noticed, which point to the disease being of infectious character. Among these are dark, discoloured, uncoagulated blood, a clay-coloured liver, opaque swelling and fatty degeneration of the internal organs, such as the heart, widespread haemorrhage, catarrhal affection of the intestinal canal, particularly of the abomasum in lambs, etc.

**Symptoms.**—The signs of cerebro-spinal meningitis are a combination of those of brain inflammation and inflammation of the spinal cord. They consist then mainly in stupor and somnolence (coma), signs of brain-irritation being rarer. These are complicated with fits of spasms in the muscles of head, neck and extremities, with a stiff, constrained attitude of the neck and a cramp-like twist backwards of both head and

neck (opisthotonus) ; there is also generally a high infective fever. When the medulla oblongata is more especially attacked we often, though not always, perceive paralysis of the muscles of tongue and pharynx, with difficulty or even complete paralysis of swallowing (acute bulbar paralysis). This arises from affection of the motor nerve-centres. At times an attack begins with symptoms of acute gastro-intestinal catarrh, with slight colic. The morbid aspect varies somewhat considerably with the intensity of the disease, and according as the brain, medulla oblongata, or spinal cord be attacked, and finally, according to the kind of animal.

i. In **horses** the disease begins sometimes with violent shivering, which may even shake the animal to and fro in its stall ; at others with extreme heaviness and somnolence, so that it staggers or falls down ; and in general the signs of a brain-inflammation are manifested, though mainly those of depression. In other cases slight colic and difficult swallowing are the signs first noticed ; the animal makes repeated and vain attempts to swallow its accumulated saliva, which eventually flows from its mouth in long threads. In rarer instances the disease commences with opisthotonus. Later on we observe stiffness of the neck and pain on touching that part ; and in lifting the head or pushing the animal back, we notice a constrained posture of the neck or even of the entire spine, attacks of opisthotonus and trismus, heat of the head and neck, with marked prominence of the muscles of the latter, which are tight and hard to the touch. The animal grows more excitable, and suffers much from hyperæsthesia and timidity. Then spasms of the facial (the face looks as in tetanus) and shoulder-muscles appear, and also of the extremities, lasting for minutes and followed by extreme relaxation, general muscular tremors and much debility. Clonic spasms of the lip and eye muscles are also observed (nystagmus). Paresis and even paralysis of the hind-quarters may occur later, with a tottering, clumsy gait and at last decided lumbar paralysis. Moreover, paralysis of the optic nerve (amaurosis), of the lips, ears, etc., may be seen. The visible mucous membranes are either much reddened or else turn yellow ; appetite is lessened or suspended, being very often complicated with difficult or paralysed deglutition and much secretion of saliva. The urine contains albumen and sometimes red blood-corpuscles ; even pronounced haematuria has been observed. The tempera-

ture may rise to  $106^{\circ}$  Fahr.; but in some cases is hardly raised at all. The pulse seems sometimes to be very frequent, at others normal, or below, being generally small and feeble.

The course of the disease is acute; its average duration being 8 to 14 days; but death may occur within 3 to 5 days. It appears to be especially violent at the beginning of an epizootic outbreak, when, indeed, sudden deaths have been noted, and the animals collapsed quickly in spasms (opisthotonus) and speedily died with the symptoms of general paralysis. In other cases the condition remains for weeks unchanged, or leads to sleepy staggers (Uhlich). Relapse is also said to be frequent. The prognosis is very unfavourable, and, according to Hartenstein and Wilhelm, the mortality reaches 85 to 90 per cent.

2. In sheep certain premonitions, such as melancholy, dejection, great dulness of the sensorium, salivation, sucking-spasms in the lips, rotary movements, etc., announce the disease itself. Soon after these the animals lie helpless on the ground, unable to rise, are frightened when touched and have various kinds of spasms. In particular the head and neck are twisted upwards and backwards so that both are in a line, or the head almost touches the withers. Chewing-spasms, grinding of the teeth, nodding the head, with twitching and convulsions of the muscles of the extremities also set in. Brow and skull are hot, the conjunctivæ much redder, the pupils dilated. In lambs the temperature is often above the normal (possibly a fatal sign). The course is sometimes very acute, so that death may occur in a few hours or days; sometimes the malady lasts one to three weeks. The usual result is a fatal one, not only with lambs, but also with older animals. Thus Schmidt records 41 deaths among 43 sheep of various ages.

3. In dogs we perceive at first signs of great stupefaction; those of delirium are rarer. The skull feels hot, the animals tremble, are feverish, show difficulty of swallowing, knock against things, stagger and even perform involuntary actions (running in a circle, rolling). The muscles of the neck are hard and prominent, the neck itself is spasmodically drawn backwards, and there may even be general cramps over the whole body, only broken by short intervals, which lead eventually to exhaustion and death. It is said that 2 or 3 days before

death the cornea is slightly dimmed. The average duration of the disease is 9 days (3 to 20); but the complaint is very rare among dogs, only 10 cases being reported from the Berlin dog-hospital in 9 years, out of 70,000 treated.

4. In cattle the disease is accompanied by disturbance of appetite and rumination, restlessness, excitement, shaking and elevation of the head, stupor, stretching the muscles of the neck, attacks of trismus and opisthotonus, spasms in the thighs, twitching the lips, rolling the eyes and collapse when the head is lifted; finally a condition like paralysis sets in. The symptoms are the same also in goats.

**NOTE.**—It is very difficult to say whether the disease of cattle described by Vogel and others as epizoötic spinal meningitis can be regarded as identical with this cramp of the neck, or even as related thereto. Its symptoms are great flow of saliva, difficulty of chewing and of swallowing, restlessness, skipping to and fro, frequent lying down and getting up, paralysis of the tongue and pharyngeal muscles, quickly developing debility, inability to rise and death after three to five days; but these can only indicate an affection of the medulla oblongata (acute bulbar paralysis); no psychic disturbances, spasms or twitchings were observed. "Meningitis spinalis boum epizoötica" certainly represents no uniform disease, for probably poisonings, especially those of mycotic character, and other diseases have been confused therewith. Thus the case described by Zipperlen evidently corresponds with mycotic intestinal inflammation (as Zipperlen himself suspected), because on dissection an extreme inflammation of stomach and bowels was discovered. See the chapter on mycotic inflammation of the bowels (Vol. 11., p. 169), which is also accompanied by much salivation, disquiet, paralysis of the muscles of tongue and pharynx, severe debility, etc.

**Progressive Bulbar Paralysis.**—Under this name we understand in man atrophy of the motor nerve-nuclei of the medulla oblongata, especially those of the hypoglossus, facialis and vago-accessorius ("Bulbus" is the old name for the medulla oblongata), in the course of which paralysis and atrophy of the muscles of tongue, lips and jaws takes place, and also of those of the face, palate, larynx and pharynx, showing itself in difficulties of speech, mastication and deglutition, increased secretion of saliva, diminished reflex excitability, etc. Pneumonia (due to foreign bodies) is also noticed as a result of the impaired power of swallowing. Degive, Gérard, Laridon and others have observed a quite similar condition in horses in North-West Flanders. They reported a gradually increasing paralysis of the tongue, lip and jaw muscles, with disturbance of appetite, chewing and swallowing (the latter finally becoming impossible, food accumulating in the mouth or falling out of it, salivation), along with emaciation, especially of the masticatory muscles, reduced reflex irritability, etc. The course of the complaint was very slow, death not taking place for six to twelve months. Sometimes pneumonia also occurred, due to foreign bodies. Treatment was fruitless. Dissection

revealed discoloration, atrophy and fatty degeneration of the tongue and lip muscles, a slight loss of volume in the roots of the bulbar nerves ; the medulla oblongata itself appeared normal (?). In the motor nuclei of man we find degeneration of the ganglionic cells with proliferation of connective tissue.

**Differential Diagnosis.**—1. Cerebro-spinal meningitis in horses is most frequently mistaken for a simple *brain-inflammation*, which is easily done, as the latter is also accompanied by spasms and twitchings. A distinction can only be drawn between them when the signs of spinal affection are distinctly marked and become prominent. 2. A differentiation between cerebro-spinal meningitis and *tubercular basilar meningitis* is also not always easy ; but is based upon proof of other tuberculous symptoms (tuberculosis of lungs or udder, emaciation), and upon the usual enzoötic appearance of cerebro-spinal meningitis. 3. *Fungoid poisoning*, especially as it occurs after eating *ustilagineæ* and mould-fungi, also gives rise to occasional mistakes. Examination of the food supplies the key here. 4. In dogs, *rabies* and *distemper* have chiefly to be considered ; but in by far the majority of cases a study of the collective symptoms will prevent any error. 5. In sheep confusion may arise with *cænurus* or *staggers*, which also appears enzoötically, though the symptoms of the latter are essentially different, but autopsy is always the surest method of diagnosis. 6. *Tetanus* is distinguished from cerebro-spinal meningitis by the absence of abatement of the spasms, and by the cerebral symptoms.

**Therapeutics.**—The treatment of cerebro-spinal meningitis is in general the same as for inflammation of the brain. Ice-bandages or cold douches on the head must be applied first of all ; derivation to the bowels by laxatives is also advisable. Then the anti-spasmodic remedies may be applied (subcutaneous morphia injections, bromide of potash, chloral hydrate, sulphonal, chloroform). Wilhelm obtained temporary relief by venesection. Johne observed a favourable result in a horse after using pilocarpine (9 grains per dose) ; but others have tried the remedy in vain. A wise precaution would be to disinfect the stable on the enzoötic appearance of the malady and to remove at once all sound animals.

**INFLAMMATION OF THE SPINAL CORD AND ITS MEMBRANES.  
MYELITIS AND MENINGITIS SPINALIS.**

**Etiology.**—These two diseases, which are of rare occurrence, have not yet been sufficiently investigated, either from their clinical or pathological aspects, and in our domestic animals can only be separated anatomically but not clinically. Their causes are chiefly to be sought in traumatic influences. Blows, shocks, or strains upon the spine, vertebral fractures and the drawing of too heavy loads are their most frequent sources. The occurrence of a suppurative myelitis, caused by a swallowed darning-needle working its way into the spinal cord, has been recorded. Rheumatic injuries have also been blamed. Inflammation may spread from some adjoining area and affect the spinal cord secondarily, e.g., from a carious vertebra. Finally, a secondary inflammation of the spinal medulla may take place in various infective diseases, such as septicaemia, pyæmia, strangles, distemper, dourine, etc. Comény, Schmidt and others have reported upon a primary infectious spinal meningitis which broke out enzootically among cavalry horses. Chronic inflammation of the spinal cord is most frequently observed among horses and dogs (so-called loin-weakness, ataxy, tabes dorsalis). Of 70,000 dogs treated in Berlin during 9 years, there were 96 cases of this ailment.

**Pathological Aspect.**—This agrees essentially with that found in brain-inflammation. In lepto-meningitis spinalis the pia mater and arachnoid are found hyperæmic, swollen and dull, infiltrated with ecchymoses, and with a serous (hydrorhachis) or purulent exudation between the spinal cord and the dura; the upper surface of the former being sometimes oedematously and suppuratively infiltrated. In traumatic inflammations this purulent exudation sometimes appears as an abscess and gives off a foul odour. The myelitis, that is, the inflammation of the spinal cord itself, is usually circumscribed and local, as in encephalitis, consisting of a purulent infiltration of the marrow with softening and haemorrhage (red softening), whereby its substance is changed into a greyish-red, pulpy mass. Abscesses are less frequent. During a chronic course new growths of connective tissue are formed between the ganglionic cells and nerve-fibres, with atrophy of the nervous elements (myelitis interstitialis,

or sclerosis of the spinal marrow). Pachy-meningitis spinalis is usually suppurative; during chronic course of the same small plates of cartilage and even of bone are sometimes found lodged in the dura mater spinalis, in the form of insular incrustations (pachy-meningitis chronica ossificans). More exact histological researches have been conducted by Dexler and Hamburger in dogs and horses suffering from spinal disease.

**Symptoms.**—The phenomena of inflammation of the spinal cord are easily explained by the functions of that organ. As is known, it forms the channel for the motor (anterior cornu) and sensory (posterior cornu) nerve-fibres of the trunk and extremities, and contains, moreover, certain centres for the bladder, rectum and genital organs. In correspondence with these facts, inflammation of the cord is accompanied by the following symptoms.

1. *Disturbances of mobility*, accompanied by signs partly of irritation and partly of paralysis. The animals show a constrained gait, stiffness in the spine, twitchings and spasms, sometimes also permanent contractions of the extensors and adductors of the hind legs (spasmodic spinal paralysis); or they stagger in walking, tumble down and are either partially (pareisis) or completely disabled (paralysis). In the latter case they cannot rise; larger animals lie altogether and can only be lifted up, while smaller ones drag themselves along (when only the hinder part is affected), drawing the hind limbs like a dead appendage behind them (paralysis of hind limbs). No movement can be produced in them by strong electric shocks, or doses of strychnine sufficient otherwise to kill. When the lumbar medulla is affected, then only the hind legs and tail are affected, but when the cervical medulla is diseased, all four limbs are struck with paralysis. In the latter case the pupil also is sometimes contracted.

2. *Disturbances of sensation*, either in the form of intensified (hyperesthesia) or of diminished or suspended sensibility of the skin-nerves (anaesthesia). The former manifests itself in pain, excitement and unrest when touched, especially near the spine. Smaller animals, such as dogs, shriek, larger ones shrink back, even try to bite, or rear wildly up (also once noticed in a horse). Sometimes every movement is marked by signs of pain. The tendon-reflexes can be produced with striking ease (Westphal's sign). Thus, if we tap a dog quite gently with a somewhat jerky movement of the finger upon

the patellar ligament, short, rapidly following flexor and extensor movements of the dog's leg will take place, with violent, convulsive twitching of the entire limb (Dexler). Paralysis of sensation is recognised by the loss of reflex sensibility to irritants, whether mechanical, thermal, or electric. Pin-pricks, intense heat and strong electric currents are none of them felt.

3. *Disturbances in the bladder and rectum.* These commence usually with retention of urine and accumulation of faeces in the rectum, resulting from paralysis of the detrusor urinæ and the muscles of the rectum. The collection of urine in the bladder leads later to catarrh, pyelitis and even nephritis. Later we find the contrary, viz., involuntary passage of urine and faeces, due to paralysis of the sphincter vesicæ and ani (*incontinentia urinæ et alvi*). In male animals we also observe sometimes continuous erection (*priapismus*) of the penis, followed later by impotence and paralysis.

In addition to these symptoms we often find at first a higher temperature in the parts affected, followed afterwards by a lower; sometimes also oedematous swelling, outbreaks of sweating, and also, in most cases of long duration, muscular atrophy. The prolonged lying of the animal soon brings on bed sores. The course is generally chronic, but the animals are usually slaughtered before the fatal end is reached. Cure is rare, or else there remains at least a permanent weakness of the loins and ataxy.

**Differential Diagnosis.**—Inflammation of the spinal cord seems to be most frequently confounded with rheumatic haemoglobinæmia, which, indeed, some look upon as an affection of the spinal medulla. The aspect of myelitis and meningitis spinalis as described in most text-books is therefore nothing else than the complaint formerly known as "black disury." The "rheumatic" paralysis of the hind limbs which appears during muscular rheumatism, and especially that form of it so prevalent in dogs as lumbago, is certainly often mistaken for inflammation of the spinal cord, as also are spinal paralyses due to pressure by haemorrhage and new growths, as well as the phenomena of paralysed hind-quarters which are sometimes seen in the course of inflammation of the kidneys, and they are not always very easy to distinguish. But the last-named complaint may always be recognised by the characteristic change in the urine; as the rheumatic haemoglobinæmia may

also be known by the persistent haemoglobinuria, the previous history of the case and its very acute course; and finally, a muscular affection is distinguished by the absence of complete disablement, and the local painfulness of the muscles affected. The differential diagnosis of inflammation of the spinal cord is more difficult in the case of other diseases of that organ, especially haemorrhage and new growths. In this respect all definite indications are often lacking, and autopsy alone can settle the question.

**Therapeutics.**—In recent cases the treatment of inflammation of the spinal cord has consisted in the application of cold or Priessnitz fomentations along the course of the spine. In many cases good results are obtained by the prompt use of skin-irritants in the form of blisters. For these we apply in the case of horses cantharides ointment, tincture of cantharides, croton oil or mustard oil; and for smaller animals cantharides-collodium is very good, as is also a salve of tartar emetic. Derivation to the bowels by laxatives is also advisable at the outset; and later, when paralytic symptoms become prominent, electricity may be usefully applied. For this purpose an intermittent (induction) current is generally used. We generally employ for dogs and small animals Spamer's apparatus, placing the electrodes in various positions at both ends or at each side of the spine, or alternately one at the toe-end of the disabled leg, the other in the middle of the spine. The strength of the current must be regulated by the degree of paralysis, and one should avoid giving too much pain. We have obtained improvement in numerous cases of chronic inflammation of the spinal cord by continuous electrification. For the motor paralysis strychnine may be administered internally. The subcutaneous dose of strychnine nitrate for a horse is from  $\frac{1}{2}$  to  $1\frac{1}{2}$  grains; for a cow, 1 to 2 grains; and for a dog,  $\frac{1}{10}$  to  $\frac{1}{5}$  of a grain. When the presence of a larger quantity of exudation in the spinal canal is suspected, pilocarpine may be tried (3 to 8 grains for a horse), and also arecoline ( $\frac{1}{2}$  to  $1\frac{1}{2}$  grains for a horse). In very obstinate cases cauterisation of the skin along the spine is recommended by some. Larger animals should also be placed betimes in slings, to prevent decubitus. Attention must moreover be regularly given to the emptying of bladder and rectum, by compression or use of the catheter in the former case, by hand in the latter. Finally, one may apply for the progressive atrophy of the muscles, and as a

substitute for electricity, irritant embrocations of spirits of camphor or of mustard, oil of turpentine, liniments of ammonia, etc., and also use massage.

**Infectious Paralysis of the Spinal Cord (congestion) in Horses.**—Under this name Comény has described an enzootic among French cavalry horses. Its chief symptom was a paralysis of the hind-quarters, which ran both peracutely, acutely and subacutely, but only in the former case was conjoined with fever. Autopsy revealed congestive hyperemia of the membranes of the spinal cord, as well as hyperemia, and softening of the cord itself. Investigations into the cause of the outbreak proved fruitless; but its contagious character was clearly proved. Schmidt has also described a similar enzootic among horses.

#### HÆMORRHAGE AND NEW GROWTHS IN THE SPINAL CANAL.

**Etiology.**—Apart from the pressure exerted upon the spinal cord by an effusion of inflammatory fluid, compression may be caused by hæmorrhage on the one hand and by new growths on the other. Of the latter, which, it is true, are very rare, the following have to be considered in our domestic animals: sarcoma of the dura (melanoma, spindle-celled sarcoma), lipoma, actino mycoma, glioma, myxoma, etc., and coenurus-cysts. Moreover, exostosis of the vertebral column, disease of the inter-vertebral joints, ossifying pachy-meningitis, etc., may all lead to compression of the cord. Hæmorrhage is caused either by traumatic influences (fracture or shock to the spine, shot wounds, etc.), or by vascular disturbances, such as emboli. The blood is found either beneath the dura or the arachnoid or in the centre of the cord itself (hæmato-myelia).

**Symptoms.**—The pressure-paralyses caused by *hæmorrhage* in the membranes or cord are characterised by their sudden, apoplectiform occurrence (spinal apoplexy). By this they are distinguished from those arising from new growths, as well as from inflammation of the spinal cord. The history generally points to some traumatic cause, or examination will discover one, e.g., in dogs, great painfulness of the spine and lumbar region when bent or stretched. Moreover, an improvement, or even recovery, may set in during the course of the paralysis, owing to resorption of the extravasated blood, which is never the case with new growths. Signs of irritation sometimes precede those of paralysis (twitchings, pain). Thus Kammerer observed a genuine attack of cerebro-spinal meningitis in a cow after hæmorrhage near the medulla oblongata. Schlesinger

saw tetanic spasms of the extremities and muscles of the back in a dog, which quickly spread to those of the breast and neck, and also clonic convulsions of single groups of muscles, sensibility to pressure and stiffness in the spine, extreme hyperesthesia of the skin, and spontaneous evidences of pain.

Compressive new growths only lead gradually to paralysis, and generally after previous symptoms of irritation. It is at first only partial (paresis), but later becomes complete (paralysis). Cure is quite impossible. The aspect of the malady closely resembles that of inflammation of the spinal cord.

**Differential Diagnosis.**—In addition to the above narrower distinction between the various spinal paralyses (caused by new growths, inflammatory and apoplectic), which is based upon their gradual or sudden appearance, the history of the case or its curability or the possibility of improvement—which distinction only too often cannot be made—it is particularly important from a practical point to be able to tell a spinal from a cerebral paralysis. We possess the following data to guide us in so doing.

1. Spinal paralyses are mainly paraplegia, and the cerebral are hemiplegia. The explanation lies in the fact that, owing to the smallness of the spinal cord as compared with the brain, the pathological process generally attacks it in its entire diameter. For the same reason the paralysis is both of motion and sensation (anterior and posterior horns or cornua); consequently all muscles situated behind the affected spot in the spine are paralysed.

2. In spinal paralysis there is usually no psychic disturbance, nor any participation of the cerebral-nerves, which signs are mostly present in the cerebral form.

3. A simultaneous paralysis of bladder and rectum points to a spinal origin.

4. Spinal paralysis sometimes advances forward, is therefore one which ascends.

5. Disturbed nourishment in the affected parts (muscular atrophy) is so far a characteristic of spinal paralysis as that the ganglionic cells of the motor anterior horns of the medulla possess a nutritive influence upon the muscles which they innervate, which is not to such an extent the case with the motor brain-centres. But muscular atrophy also occurs in cerebral paralysis (trigeminus).

Compressive paralysis in dogs is not unfrequently confused

with a spinal paresis or paralysis caused by heart-disease (valvular defects). Thus we sometimes observe paralysis of the hind quarters during incipient dilatation of the heart following on valvular defects, which arises from venous hyperæmia (or arterial anæmia) of the medulla spinalis, or in other cases from thrombosis of the femoral arteries which has occurred in the course of such heart affection. In every case then of spinal paralysis in a dog it is well carefully to examine the heart.

**Therapeutics.**—The treatment of paralysia due to pressure is the same as for inflammation of the spinal cord. That from new growths is incurable. In haemorrhage of the spine one might administer internal laxatives or iodoform to assist resorption.

**Ataxy.**—By "ataxy" we understand a disturbance of the co-ordination of movement, whereby all the muscles needed for a particular action are not set in motion at the right moment, but some either too soon or too late; or some groups of muscles alone are moved (such as the flexors) without any corresponding action of the extensors. One of the commonest ataxies is the "cock-stride" of horses, which may in many cases be a result of spinal disease. Atactic movements caused by an interstitial myelitis (pressure-atrophy of the nervous elements by excess of connective tissue) have been described in horses by Weber and Barrier. In one case of cock-stride Bolton found chronic degeneration of the lower dorsal and lumbar medulla on scattered spots and in the form of swelling, disintegration and diminished power of taking on stains in the axis-cylinders, disappearance of the medullary sheaths and a formation of interstices in the supporting substance.

**TROTTING OR NIBBLING DISEASE IN SHEEP (*Trotters or Nibblers*).**

**Definition.**—This disease is a chronic, non-febrile hereditary affection of the spinal cord, the nature of which has not yet been sufficiently investigated. It consists mainly in increased sensitiveness (hyperesthesia) and in weakness and signs of paralysis in the hind quarters, with extreme emaciation, leading constantly to death. The comparison often made with tabes dorsalis (grey degeneration of the posterior columns) of man rests upon pure hypothesis, though it has some ground of probability. As we have no exact scientific details of the pathological lesions of the spinal cord, we must confine ourselves to a brief discussion of its occurrence, etiology, symptoms and prophylaxis.

**Occurrence.**—The trotting sickness was almost unknown before the introduction of the nobler breeds of merinos (imported in 1763 from Spain), but has since become widely spread. It is particularly common in the very fine Electoral herds, while rarer among the commoner sheep of the country or the Negretti breed. The males sicken most readily, especially young rams from two to three years; females and wethers less frequently. The disease, which is one of the worst that can attack a flock, is therefore much commoner in Prussia and Saxony, with their finer sheep-breeding establishments, than in South Germany.

**Etiology.**—The causes of trotting sickness are first to be sought in heredity. It is usually introduced into a flock by a breeding ram, which is already visibly affected or else bears in it the germs of the disease. False principles of breeding exercise great influence; exaggerated refinement of the race by too little freshening with new blood, exclusive breeding between nearly related animals, pampering the young, unhealthy forcing, attempts to hasten maturity, the use of ewes when too old for breeding and the sexual over-excitement of males by too frequent and too early copulation. Whether, besides these causes, others, such as climatic or local influences (swampy, flooded pastures with too luxuriant and rich fodder, or very meagre, sandy soil), co-operate cannot at present be decided.

**Symptoms.**—The first stage of trotting sickness opens with signs of no apparent importance. The animals are very timid, behave shyly and hurriedly, tremble when seized, have an unsteady, staring and stupid look, nod with their heads, also bend them backwards and display a peculiar trembling motion of the ears—the latter especially when the sun is very hot. This stage lasts 4 to 8 weeks in winter, but only 8 to 14 days in summer.

The second stage has a winter duration of 2 to 4 months, and in summer lasts as many weeks. It also begins gradually and with general slackness of the muscles and growing weakness in the hind limbs. The animals have an insecure, tripping or trotting gait (hence the name), in which they straddle with the hind feet and thrust them far under the body. Movements resembling the step of fowls are seen ("cock-stride"). The power of galloping and coition is lost. This ataxy is succeeded later with very severe itch, especially in the lumbar region,

which is betrayed by much gnawing and rubbing (hence the name of nibbling sickness). The animals bite themselves on the root of the tail, loins, back and also on the hind, and even the fore legs. This produces bald, inflamed places upon the skin, or else the wool is rubbed off, becomes rough and shaggy. Paralytic signs, which travel forwards, appear during the later course in the hinder limbs, so that the sheep stagger about when walking, tumble down, can scarcely rise and grow very thin. Death then follows, preceded by increasing debility and exhaustion.

**Differential Diagnosis.**—This disease may be confused with rump-staggers (coenurus-cysts in the spinal cord) and other spinal paralyses. A distinction must be based upon the symptom which is particularly important in trotting sickness, viz., the presence of timidity and nibbling. The occurrence of the disease chiefly among male animals and in finer breeds is also evidence of trotting sickness.

**Therapeutics.**—Treatment of the sick animals is as good as useless, and it is best to slaughter them early. Much more important is prophylaxis, which consists in preventing all affected animals from breeding, in not using the rams until after their second year and avoiding any sexual over-excitement; also in natural methods of keeping and feeding, especially of the young. It is also well to avoid too great a refinement of race, and therefore not to breed from animals too nearly related.

**Tabes dorsalis.**—Under the names of tabes dorsalis, sclerosis of the posterior spinal processes, or progressive motor-ataxy, we describe in human medicine a chronic affection of the spinal cord accompanied by signs of paralysis, the causes of which are not yet precisely known. The former erroneous opinion traced them to sexual excess. It seems that severe chill and great over-exertion of the legs, on the one hand, and an inherited predisposition on the other, are the most important sources of the disease. In regard to the latter point, it has been noticed that several members of the same family have suffered from tabes, which attacks men most frequently of from thirty to fifty years. The malady consists anatomically in grey degeneration of the hinder processes of the spinal cord, with extreme proliferation of connective tissue (sclerosis). The symptoms are in the main those of disturbed co-ordination of muscular movement (ataxy). They begin with signs of spinal irritation, shown in hyperesthesia of the skin and in keen and darting neuralgic pains in the arms and legs, which stage may last for years. The ailment is often confused with rheumatism, ischias and other independent neuralgias. The

second and so-called manifest stage consists in derangement of co-ordinated motion (motor-ataxy). It begins in being easily tired, in a certain insecurity and awkwardness of gait, whereby the feet are often lifted unnecessarily high, moved backwards or set down with a stamp ("cock-stride"). Sensibility is at the same time weakened, so that the resistance of the ground cannot be felt ("walking on clouds"). The third, or paralytic or paraplegic, stage is that of complete spinal paralysis.

Does this tabes dorsalis of man exist among animals? This question can only be tentatively answered. The trotting sickness of sheep, for instance, is very like it. Compare their heritability, their occurrence chiefly in males, their two stages of irritation and paralysis and the undoubtedly motor-ataxy. We believe also that in treating dogs for ataxy we have several times had true cases of tabes dorsalis before us, but have made no pathological investigation. Hamburger, on the contrary, proved decided tabes-like degeneration of the posterior processes in a dog, without, however, having seen the dog alive. The proof of tabes dorsalis in domestic animals during life is so difficult, because the subjective symptoms (hyperesthesia, neuralgia, loss of feeling) are not usually well marked. But it is much to be desired that more exact pathological investigation should be made, combined with observation of the symptoms during life, viz., in sheep (trotting sickness), in dogs (ataxy) and in horses (cock-stride, lumbar weakness).

## CHAPTER XVII.

### DISEASES OF THE PERIPHERAL NERVES.

#### PARALYSIS OF INDIVIDUAL PERIPHERAL NERVES.

**Etiology.**—The causes of paralysis of the peripheral nerves in our domestic animals are usually traumatic influences acting upon some particular spot in the course of such nerve. Such are contusion of the facial near the temporo-maxillary joint at the place where it bends round the inferior maxilla ; pressure exerted by tumours on the course of a nerve ; injuries and lacerations of the supra-scapular when running violently against some hard object, in collisions or when lying on the ground. But central disorders may be responsible for such paralyses, viz., tumours, abscesses and inflammation on the base of the brain. Rarer causes seem to be chills, frights, etc. Finally, infectious diseases, especially distemper of dogs, may cause peripheral paralysis, as may also poisoning.

**Symptoms.**—These are merely disturbances of function in the individual nerves affected.

i. **Paralysis of the Facial** (7th) is seen in disablement of the facial muscles on one or both sides of the head. When on one side only, the upper lip and tip of the nose are drawn towards the sound side, the lower lip hangs limply down or is also twisted to the contrary side ; food gathers between the cheeks and teeth ; the eye on the paralysed side can either not be shut at all, but stands wide open and weeps (peripheral facial-paralysis), or the affected eyelid droops over the eye (ptosis), so that it appears to be shut (central facial-paralysis). As a consequence of one-sided action of the nostrils, dyspnoea may occur on any severe exertion. The course depends on the degree of paralysis. Recovery is not rare.

2. **Paralysis of the Trigeminal** (5th) causes first, one-sided anaesthesia of the face, oral cavity and tongue, also of the eye, while the insensibility of the latter brings with it the non-removal of irritant bodies, with consequent inflammation and ulceration of the cornea. 2. When combined with paralysis of the motor branch of the trigeminal, it causes also disablement of the chewing muscles, with difficulty in feeding, lodgment of fodder in the mouth, salivation, hanging-down of the lower jaw, irregular attrition of the molars and atrophy of the muscles of the jaw. This latter form of trigeminal-paralysis occurs particularly in dogs during rabies and distemper.

3. **Paralysis of the Supra-scapular** leads to true or neuropathic paralysis of the shoulder. It is characterised by atrophy of both the serratus muscle and the rotator of the shoulder, as well as by a peculiar lameness of the shoulder, in which, when pressure falls on the foot in question, the shoulder bends outwardly, so that a varying space is left between the thoracic wall and the elbow. Contrary to the conditions in displacement of the shoulder caused by laceration of its muscles, there is here no inflammation. The paralysed muscles do not react to an electric shock. Prognosis is not unfavourable.

4. **Paralysis of the Radial**, which innervates the extensor muscles of the fore-arm, especially the triceps, as well as those of the lower joints, reveals itself in inability of the affected fore-leg to support the body. The leg collapses at every joint, because they cannot stretch to resist pressure, but by pressing against the knee it is able to support the body. The paralysed muscles feel limp and, if the condition last long, become atrophied. Prognosis is mostly favourable ; and this form of paralysis in a horse is usually cured in six to eight weeks.

5. **Paralysis of the Crural** affects the extensors of the patella ; consequently here also, on weight being thrown upon the limb, there is abnormal flexion of the stifle-joint, because an extension of the same has been rendered impossible by the paralysis. If this last for long, the muscles of the patella become atrophied.

6. **Paralysis of the Ischiatic** results in complete disablement of the muscles of the affected hind-leg, with exception of the extensors of the patella, which are innervated from the

crural. Such muscles soon become atrophied and lose their reaction to electricity.

7. **Paralysis of the Tibial** is shown in an abnormal flexion of the hock-joint and collapse of the foot as soon as weight bears upon it; the paralysed gemelli muscles, as well as the flexors of the foot, feel slack to the touch and are unable to extend the hock. Consequently the gait is clumsy, the lower leg is stiffly held, movement is like that of a hen walking, and the feet are lifted very high. The peroneus is, however, not paralysed.

8. **Paralysis of the Obturator** means also paralysis and atrophy of the adductors of the hind thigh (gracilis, sartorius, pectineus, obturator, adductors), with abduction of the leg and incapability to move it inwards.

**Treatment.**—This consists in massage and application of irritant remedies, of cold in the form of douches, of electrical currents, blisters, subcutaneous injections of veratrine and strychnine, of the cauterizing iron, etc.

**Neuralgias**, which are paroxysms of pain in the region of distribution of any particular nerve-branch, have not yet been observed in our domestic animals, but probably only because they lack the subjective power of conveying their sensations to us. In man they are somewhat common, and attack especially the trigeminus, occipitalis major, the brachial and lumbar plexi, the intercostal nerves and the ischiadicus (neuralgia of the latter is called "ischias"). Friedberger noticed in a horse a peculiar and exceedingly severe itching on the skin of the neck and forearms, for which no local causes could be discovered, but which was considerably relieved by subcutaneous morphia-injections. Whether this evident neurosis of sensation was of neuralgic nature, or is to be looked upon as corresponding to the pruritus of man, cannot be decided. Strebler has described a similar case in cattle. Serini believes that he has seen ischias in dogs and cattle, and Giovanni a neuralgia of the brachial plexus in a horse.

**Facial Spasms** (*Tic convulsif*) in a dog, and in the form in which they occur in man, have been described by Cadiot, Gilbert and Roger.

**Spasms of the Accessorius** in a horse are described by Aruch.

## CHAPTER XVIII.

### NEUROSES WITHOUT KNOWN ANATOMICAL BASIS. EPILEPSY.

**Definition.**—Epilepsy is not very common among our domestic animals, yet occurs in them all, including poultry. It is a chronic brain affection with fits of disturbed consciousness and sensation and accompanying muscular spasms, between which are free intervals of varying periods. Regarding the nature and seat of the disease recent investigations have procured us some knowledge, which, however, is far from complete. The seat of epilepsy is especially to be sought in the motor centres of the cortex of the cerebrum. The chief experimental proof of this theory (the cortical theory) lies in the fact that by irritating the motor cortex-areas of the cerebrum, epileptiform fits can be produced in animals, and that, after such a centre has been extirpated, the spasms connected with its group of muscles cease. Moreover, the spasms in epilepsy correspond entirely with those of the muscular groups innervated by the motor-centre in question, and epileptic fits have been noticed in man after pathological affection of the cerebral cortex. But, above all, the invariable complication of the spasms with unconsciousness indicates clearly the cortex as the seat of origin of the malady. Bergmann has shown that such injuries to the skull as affect more especially the cerebral cortex produce both epileptiform and true epileptic fits. The experiments of Hitzig, Fritsch, Luccani and others have proved that such fits may be caused by electric irritation applied to any part of the cerebral cortex, but that in the motor region the very faintest shock suffices. Moreover, after removal of this motor region, no attacks can be produced electrically from any portion of the cerebral cortex; but, on the other hand, they can still be produced even though the entire cortex be removed excepting

that of the motor region. Besides the cortex of the cerebrum, more deep-seated portions of the brain seem sometimes to be the starting-point of epilepsy (infra-cortical or medullary theory).

What the exact changes in the cerebral cortex are, is as yet unknown. Anæmia of the brain caused by sudden vascular spasm was formerly suspected (Kussmaul and Tenner); but other observers could not demonstrate the presence of such an anæmia. There is, therefore, nothing else left but to assume a peculiar fleeting state of excitement in the cortex of the cerebrum, and especially of its motor cortex-centres, as the producing cause of epilepsy, which is perhaps caused by an abnormal chemical or molecular composition of the substance of the cerebral cortex (Rosenbach).

**Etiology.**—In practice it is well to distinguish as regards their causes several varieties of epilepsy.

I. *True genuine idiopathic or spontaneous epilepsy* is a purely functional neurosis of the brain, without any discoverable pathological change. Its essential causes are not known. Among those which produce a predisposition thereto, heredity plays a leading part. This has been established by various authorities. Thus La Notte noticed the frequency of these fits in a herd descended from two epileptic bulls; they usually attacked cows after their first calf, and oxen when they were set to work. For this reason epilepsy is reckoned as a "hereditary taint" in breeding paddocks. It is possible also that a bad condition of nourishment, rickets, the pampering of young animals, etc., may all predispose thereto; breeding stallions are particularly subject to attack. Out of 70,000 dogs treated during nine years in the Berlin dog-hospital there were 419 cases of epilepsy; on the other hand, it occurred only three times in two years among 30,000 service horses of the Prussian army.

Among accidental causes which may produce epileptic fits in domestic animals when the predisposition already exists, experience has taught us to recognise several. As in man, so here also, certain psychic emotions stand in the first place, such as fright, excitement, dread. Thus it is reported that a fourteen-year-old gelding, trained by its owner, had its first fit when ploughing, on being startled by a suddenly thrown white cloth. In another horse the cause was a violent fright caused by a stork unexpectedly flying up; a dog had a fit as a locomotive rapidly dashed past. In horses epilepsy often arises from glaring

rays of light acting on the eyes, or from too sudden changes from light or darkness, thus, for instance, in driving rapidly towards the sun when low down on the horizon, or in an avenue when the setting sun throws the tree-shadows diagonally across the road, also in a strong reflected light from surfaces of water or snow.

2. *Symptomatic epilepsy*, in contrast to the genuine idiopathic form, is produced by certain pathological, chronic, morbid conditions of the brain and its membranes, such as new growths, parasites (*cysticercæ* in pigs, *echinococci* and *coenurus* in cattle and sheep), abscesses, older encephalitic foci, tubercles, chronic pachy-meningitis with thickening of the dura, exostoses, etc. In its nature, therefore, this form is quite different from true epilepsy, and only agrees therewith in its symptoms; the spasms which it produces are consequently always spoken of as epileptiform, in contrast to the truly epileptic. But since the causes of symptomatic epilepsy can only be known after death, its spasms are also alluded to in life as epileptic fits. Foren-sically these symptomatic forms are included in the designation "epilepsy," from which a warranty may be demanded.

3. *Traumatic epilepsy* starts from injuries of the cranium and brain; and the epileptic fits often do not occur until long after infliction of the injury. The pathological process may therefore vary. Either we have a lesion of the cerebral cortex, a compression of it by blood, bone-splinters, etc., or a disease of the membranes of the brain. This form of epilepsy has been produced experimentally in guinea-pigs by blows on the skull, the disease setting in after some weeks (Westphal). It is also asserted that epilepsy thus traumatically caused by mutilation of the cortex of the cerebrum in dogs can be transmitted by heredity to their offspring.

4. *Reflex epilepsy* occurs in certain nervously disposed animals after the action of irritants upon their peripheral nerves. Among these are wounds and contusions of the peripheral skin-nerves, compression of the same by tumours, as well as irritation of the mucous membranes by parasites. Of the latter, *ascaris megalcephala* and mycotic otitis (*aspergillus nigricans* in the shell of the ear) are blamed in horses, and in dogs tape-worms and the pentastoma *taenioides* which occurs in the frontal sinus. Nervously disposed dogs are said to have epileptiform spasms in constipation, which may return again later. Injury to the mucous membrane of the mouth by sharp-edged teeth in horses when eating may occasionally bring on an epileptic

attack. Gerlach observed a horse which collapsed with epileptic spasms on being touched upon the withers, where it was very sensitive. Schrader saw another which fell into fits on touching a certain spot upon its skin in the middle of the left side of its neck. Brown-Séquard and others produced this reflex epilepsy experimentally in rabbits by injuries to the peripheral nerves. For instance, in consequence of section of the ischiatic the malady appeared in from 11 to 71 days, continued for some time and was occasionally even transmitted to progeny.

**Pathological Aspect.**—As already stated, there are no pathological changes in the brain in true epilepsy. But in the other forms we find, according to their respective etiology tumours, exostoses of the cranium, thickening, adhesion or ossification of the cerebral membranes, encephalitic foci, etc. Thus in an epileptic cow Voigtländer observed in the middle of the right hemisphere of the cerebrum a partial suppurative encephalitis in consequence of fracture of the horn. Cerebral oedema, chronic hydrocephalus and partial atrophy of the brain are also found. In nine epileptic horses Bassi found pronounced asymmetry of the skull. Siedamgrotzky also noticed in a dog that the skull-bones were distorted, lumpy and uneven, the cranial cavity and the brain were very small, and there was chronic hydrocephalus with dilatation of the ventricles and atrophy of the adjacent brain-parts (rickety changes).

**Symptoms.**—The characteristic epileptic fits occur either as general muscular spasms spread over the whole body and coupled with insensibility (complete, general epilepsy), or the spasms are restricted to single groups of muscles, especially of the head and neck, and during which the animals can retain their feet (incomplete, partial epilepsy). But transitions from one form to the other occur in the same animal.

I. *Severe epileptic fits* (complete general epilepsy), as observed in the stable or when using the animals, occur sometimes very suddenly. Dogs and pigs, after staggering for a moment, suddenly fall senseless to the ground, often with loud cries, and are attacked by violent tonic and clonic convulsions. But usually there are prodromal signs, premonitions, which seem to point to loss of equilibrium, or dizziness. In rarer instances the attack commences, as in man, with peculiar disturbances of sensation on single limited spots upon the body, so-called sensible "aura." Thus Hertwig saw the attack begin in two

horses by their endeavouring to scratch their heads with their right hind foot. The usual course of a fit is as follows :—Horses in the stall suddenly hang on the halter; those in movement suddenly stand still and display a certain excitement and dread. Dogs run about terror-stricken and often as though senseless, or stare around stupidly and shake their heads. In all animals the glance is staring and expressionless, the eyeball looks rigid and fixed, they begin to tremble, stagger to one side or a few steps back and straddle widely with the legs to support their tottering bodies. The convulsive twitchings which now set in generally begin at the head, in other cases in the neck, and consist in blinking with the eyelids, rolling, twisting and even squinting of the eyeball, twitching, dragging movements of the muscles of the lips, nose, cheeks, and ears. To these spasmodic action of the jaws is added, whereby the abundantly secreted saliva is worked into a lather. The jerky withdrawal of the posterior maxilla occurs sometimes so violently that loud smacking noises are heard. In other cases the jaws are tightly pressed together, we hear grinding of the teeth, or again the mouth is fixed wide open for some time. The spasms gradually pass on over the neck and trunk. Head and neck are stiffly bent in one direction, mostly to one side, the same also happens to the back, so that the head and neck may be drawn against the lateral thoracic wall. The animals consequently lose their balance and tumble down. While on the ground the convulsions attain their greatest severity and extend to the extremities. The animal struggles or beats convulsively with its feet, and complete loss of consciousness and of sensation soon follows.

The mucous membranes of the head, especially the tarsal conjunctiva, are pale at the beginning of such an attack, the pupils dilated and insensible to light, and reflex processes are mostly suppressed. The heart action is irregular and slow, the speed of the pulse sinks below half its normal rate, the pulse itself being small and hard. Breathing is strained while the animal lies, even panting and rattling. Dogs and pigs often utter shrieks and squeals. In consequence of the dyspnoea and violent muscular action, considerable venous hyperæmia of the head may set in towards the end of a fit; outbreaks of sweat are also noticed, particularly over the elbows and in the flanks.

These convulsions sometimes cease suddenly, at others only gradually, and not rarely give place to a brief attack of general tetanic spasms, during which involuntary passage of faeces and

urine is sometimes seen. When the attack is over, consciousness and sensation generally return quickly. Only in very severe cases does a visible indisposition, which may last up to an hour, remain behind. The animal rises from the ground and gradually recovers, manifesting for a time a certain lassitude and exhaustion. Dogs often get up as soon as the fit is over, shake themselves and recognise their surroundings ; but sometimes the attack is ended by a deep, snoring sleep. Some have observed as the immediate sequel of such fits an unusual and even maniacal excitement. Dogs will run aimlessly away, or show a tendency to bite. Schrader saw a horse which bit its own fore-leg after the spasms had ceased.

2. *In slighter epileptic fits* (incomplete partial epilepsy), which are also accompanied by suspended consciousness, the spasms often remain localised upon certain muscles of the head, or they pass on over the neck and trunk, thus forming a transition between the slight and the severe. Frequently we notice only blinking with the eyes, squinting or rolling the eyes, with spasmoid twitching of the facial muscles, especially of the lips. Such slight attacks in horses bear a great likeness to vertigo ; they occur particularly during movement and in horses with blinkers, but may also happen in the stable or directly a horse is taken out and harnessed. In stronger spasms the animal shows a backward twitching of head and neck, which are best seen sideways, and trembling of the fore-quarters (cows fall on their knees or lean against the wall). These in addition to the spasms in the head just named.

**Duration and Course.**—The *duration* of epileptic fits is various, being generally longer in the severer attacks. It varies from a few minutes (we saw one which only lasted fifteen seconds) to a quarter or half-an-hour. The return of the paroxysms occurs at irregular intervals. They may be repeated several times in a day, or may cease for months and even for more than a year. Sometimes they are more frequent at the beginning of the disease, sometimes during its later course. The violence of the several attacks is very different, even in the same animal ; and slighter fits may continue at the same degree for many years without an increase of intensity. Between the fits, animals usually appear quite healthy ; more rarely there is a permanent psychic depression, diminished intelligence, even dulness and imbecility, trained dogs have even been known to forget their tricks. But these signs may also arise from a

simultaneously developed chronic hydrocephalus or cerebral atrophy.

The course of epilepsy is extremely chronic, the disease maintains its ground for years and even for life, an early disappearance after a few months is extraordinarily rare. It may be very easily confounded with other conditions, and yet with proper treatment epilepsy is sometimes curable. As such it does not cause death, excepting when severe injury, haemorrhage on the brain, etc., is produced accidentally when falling, which however only rarely happens.

**Differential Diagnosis.**—The diagnosis of epilepsy is mainly based upon its chronic course and upon the paroxysmal character of its muscular spasms, which are always accompanied by loss of consciousness. Simple though it may seem to diagnose the disease from the above indications, yet in practice it is not always so easy and certain. Epilepsy may sometimes be confounded with the following diseases.

1. With  *eclampsia* and *acute cerebral spasms*, the so-called epileptiform or epileptoid fits, which latter occur as symptoms of various acute brain-diseases or infectious ailments, such as distemper in dogs, or as part-symptoms in certain poisonings, or as simple and quickly passing reflex phenomena (teething convulsions). The only distinguishing characteristic here is the acute course—the spasms are exactly like those of epilepsy and are combined with loss of consciousness—but this acute course is not always easy to prove; then again, reflexly excited sympathetic cerebral spasms may also assume a chronic course, if the reflex agent continue its operation for long, as, e.g., with intestinal parasites. In the latter case differentiation is exceedingly difficult and diagnosis often impossible during life.

2. The slighter epileptic fits may also be confounded with *vertigo*. The decisive criterion is that convulsions do not occur in vertigo, but in such slight cases the convulsions may be so feeble as only to be noted on careful examination (in man they sometimes lack entirely); on the other hand, the struggling movements of the feet in very severe attacks of vertigo, when the patients fall down, are very much like convulsive spasms. This is why, from the scientific standpoint, no clear border-line has been drawn between epilepsy and vertigo.

3. *Thrombosis of the posterior branches of the aorta* (iliac, lumbar and pelvic arteries) has not at all rarely been mistaken

for true epilepsy, owing to the sudden break-down of the hind-quarters. But diagnosis is secured by examination per rectum, by the peculiar kind of functional disturbance in the hinder limbs (paresis), the absence of spasms, and the possibility of producing at any time the symptoms of thrombosis of the aortal branches by suitably moving and exerting the animal.

**Therapeutics.**—Apart from the avoidance of any known causes, one may, at the beginning of an attack, always lessen its violence and perhaps prevent the animal from falling down if we hold its head firmly, cover up its eyes and bend its neck energetically towards the opposite side from the paroxysm. Other remedies for shortening the fits, such as smelling drugs, inhalation of chloroform, sprinkling with cold water, etc., are not applicable to large animals, and in smaller ones are almost worthless.

Against the disease itself a number of drugs are in use, which are mainly effective as they reduce the enhanced irritability of the brain. Success has been attained most frequently with dogs. According to our own experience and that of others, bromide of potash and bromide of soda are particularly good remedies for dogs, we give 3 to 4 doses daily for a considerable time, viz., 8 to 15 grains dissolved in water. Various bromides can also be prescribed together in the form of so-called bromide-water: Potassium bromide and sodium bromide of each 2½ drams; ammonium bromide 1½ drams, water 10 oz., from a teaspoonful to a tablespoonful to be given three or four times daily. To horses and cattle we give this along with other salts and with aromatic vegetable powders mixed with their food, in doses of 5 drams to 1½ oz. Of other remedies we might mention: nitrate of silver (dogs, ½ to 1½ grains per day in three or four doses; horses, 15 to 30 grains); oxide of zinc (dogs, ½ to 3 grains); belladonna (atropin. sulph. ½ to ½ grain for dogs and ½ to 1½ grains for horses); sulphate of copper, valerian root, etc.

#### ECLAMPSIA:

**General Notes on the Definition of Eclampsia.**—Under the title of eclampsia a number of diseases both of man and of animals are grouped together which, owing to their manifest differences, can hardly be brought under one scientific

definition. In its widest sense the word eclampsia is used as a collective name for several convulsive conditions whose common characteristic consists in tonic and clonic spasms, which run acutely with or without unconsciousness. But under this very wide definition have been included a number of purely symptomatic spasms arising from well-known pathological changes, which do not merit the name of a special disease. Thus the cerebral spasms which occur during brain diseases, or the epileptiform spasms of distemper should not have a separate name of their own, nor those which arise as symptoms of uræmia or of certain poisonings, although at one time men spoke of an eclampsia uræmica or saturnina (lead-poisoning).

On these grounds the definition of eclampsia has recently been narrowed, and the name only applied to the cases of epileptiform spasms which, independent of certain organic diseases, manifest themselves as a pure neurosis in the form of an independent acute malady. According to this view, eclampsia would mean the same as acute epilepsy, in which the eclamptic convolution passes off exactly in the same manner (and mostly in the way of reflex movement) as the epileptic, the two being only distinguished by their acute and chronic course. If we compare the several acute convulsive conditions recorded in our veterinary literature as eclampsia with this narrower definition, we shall find that an eclampsia identical with acute epilepsy does occur among our domestic animals. To this class belong especially the convulsions which are seen in new-born and very young animals (corresponding to eclampsia infantum), and certain descriptions of acute reflex spasms in full-grown animals. On the other hand, that which is commonly known as the "eclampsia of bitches" does not seem to belong to this form, because unconsciousness is usually unaffected, whereas in acute epilepsy there is a loss of consciousness. We have, nevertheless, followed the usual custom of treating the eclampsia of bitches in this chapter, as, owing to our imperfect knowledge of its nature, it is difficult to know where else to introduce it. But there is no ground for describing paralytic milk-fever as eclampsia, or for discussing it here, as we have more fully explained under the heading of Puerperal Fever.

1. **Eclampsia of Young Animals** occurs especially among teething dogs and sucking pigs. Convulsions are observed very similar to the fits of children: twisting of the eyes, tremblings, jaw-spasms, grinding the teeth, foaming

and salivation, twitchings of the body, aimless running about, shrieking and whimpering, etc. Many animals expire suddenly with a loud cry. The causes are unknown. As a rule, a nervous disposition is blamed, or certain peripheral nervous irritations (change of teeth, helminthiasis, gastric or intestinal catarrh). According to later investigations in man, the cause is supposed to be poisoning (multiple epithelial necrosis of the liver and kidneys, according to Schmorl) or infection (basilar embolism of the liver and kidneys, according to Gerdes). Treatment is the same as for epilepsy, and consists in the use of anti-spasmodic remedies, especially bromide of potash, urethane, sulphonal, and chloral hydrate. One may also try clysters of ether, decoction of valerian root, etc.

2. **Eclampsia of Full-grown Animals** is very uncommon, and its morbid aspect most frequently that of acute reflex epilepsy. Thus, many animals fall into acute epileptiform spasms on being touched upon certain wounded or inflamed spots—cows, for instance, on touching sensitive injuries at the root of the horns. In the same way the epileptiform spasms which are observed in dogs after irritation of the external ear-passages by ear-mites must also be regarded as eclampsia. It is also not unlikely that many cases of reflex epilepsy are nothing else but repeated attacks of eclampsia, although we must not forget the possibility of the latter changing into true epilepsy. Lustig describes epileptiform convulsions in a horse which was in the convalescent stage of a severe lung-affection. Albrecht, Lameris and others several times observed eclamptic fits in cows soon after calving, which had nothing to do with milk-fever and took the form of tetanic spasms in neck and head, grinding the teeth, unconsciousness, falling down, spasmodic movements of the limbs, twisting the eyes, etc. These fits lasted from a few minutes to half-an-hour, and when over the animals were generally quite well. The shortest duration of the ailment was 2 days, the longest 18 days. Similar attacks were seen in goats. The causes are here probably to be sought in inflammatory conditions of the uterus.

3. The so-called **eclampsia of suckling bitches** is a disease whose causes are still very obscure. As it occurs mostly in bitches when giving suck, the act of suckling must as such stand in close etiological connection with it, so that we may presume an abnormal irritation, starting from the

nerves of the milk-glands, to be its cause. But more recent investigations (Friedberger, Druet) have shown that the malady does not appear exclusively just after giving birth, but may occur some days before, or even some weeks after. In one case the spasms were seen eight days before parturition and in another 50 days after. We must then here presume a predisposition to spasms arising from the state of pregnancy or from having brought forth. Those which seem most inclined to the sickness are the smaller, tenderer breeds, also pampered animals, very intelligent ones, and those which yield much milk. As accidental causes, some blame strong emotion, such as grief for lost puppies, fear or excitement on being punished, and also cold. The Berlin dog-hospital reported 64 cases of eclampsia among 70,000 dogs treated in 9 years, as against 419 of epilepsy.

The *symptoms* generally appear in the interval between the second and thirtieth day after giving birth, and consist essentially in fits of tonic and clonic spasms, especially of the extensor muscles, the sensorium remaining fairly clear. The attacks come suddenly without noticeable warning. The dog shows a certain unrest, excitement and alarm, the latter being especially manifested in its eyes; breathing is quick, short and strained, as also are both pulse and heart-beat; the visible mucous membranes of the head are reddened, and sometimes after even a quarter of an hour a striking and often rapidly increasing weakness is observed in the hind-quarters. The animal is soon unable to stand, falls down, lies on one side with her feet stiffly stretched as though dead and in rigor mortis, so that the extremities can sometimes only be bent with considerable force, and the bitch, if placed on her feet, will stand like a saw-bench. Every independent motion of the limbs seems to be impracticable. At the same time brief convulsive tremblings of the limbs and also of the trunk occur, with distinct clonic spasms, which not rarely are very violent and affect especially the extensor muscles of the limbs, neck and back, but also occur in other places, such as the temporal muscles or those of the eye. With the appearance of the spasms, breathing becomes more rapid (up to 100 a minute) and at last throbbing, panting and languishing, the mouth being wide open, often covered with foam, and the tongue hanging out. The heart is palpitating and its frequency increased to 160 beats a minute or more. Sometimes the jaws are convulsively opened and shut, the tongue drawn back and swallowing movements made. The

redness of the mucous membranes of the head continually increases, the injection of the conjunctival vessels being particularly marked; even haemorrhage in the mucous membranes may take place. The abdomen is often sensitive to pressure, eating and drinking are entirely relinquished, and also the passage of faeces and urine. After an attack the latter is found to contain albumen. At the outbreak the teats are very much swollen, very full of milk and high in temperature, but later on are mostly quite collapsed and empty. Consciousness and sensibility are in no way disturbed. The face, as Mauri very justly remarks, manifests fear rather than pain. The pupils are of normal size. External irritants cause no noticeable increase of the spasms.

The course of the disease is acute, often even peracute. The attacks generally last one to two days, more rarely only a few hours, the spasms do not continue with equal violence, there being intermissions during which the animals try to stand up, which, however, they can only do with the fore legs. Finally, the convulsions give place to a sleepy condition. If this be then left to itself, death generally follows in one to two days, from paralysis of the brain or asphyxia. But proper treatment, on the contrary, regularly leads to recovery, usually in a very short time and even in an hour. We have occasionally seen instances of spontaneous recovery, but they were very rare.

A confusion of the eclampsia of suckling bitches with strychnine poisoning is scarcely possible. The absence of an increased reflex irritability of the spinal cord, i.e., reaction to external irritation, which absence marks this form of eclampsia, makes differentiation easy. It may also be known from epilepsy by its acute course, and from distemper by the absence of the other characteristic signs.

Treatment consists either in subcutaneous morphia injections (Siedamgrotzky), an average of  $\frac{1}{2}$  grain of morphium hydrochloride in 15 minims of water, or in cautious chloro-forming up to the commencement of narcosis. We have also observed good results from urethane (1 $\frac{1}{2}$  to 5 drams) and hypnone (4 to 30 grains). Mauri speaks well of chloral hydrate (8 to 30 grains); Trasbôt of ether (ether-syrup); and Zimdel recommends chloroform-syrup (15 grains of chloroform to 3 oz. of syrup, a teaspoonful to be given four times at intervals of 15 minutes, and then after intervals of two hours). Possibly the favourable effect of these narcotics, which also dilate the

vessels, may give us some hint as to the nature of the disease and allow us to trace it from a rapidly-developed and just as rapidly-corrected disturbance of circulation in certain cramp-centres. Marked pathological lesions are not present, and cannot be found by dissection. Possibly the ailment springs from anaemia of certain nerve-centres in the brain, set up reflexly by the nerves of the mammary glands or of the uterus. This theory is supported by the fact that in many cases of eclampsia we have been able to demonstrate by the ophthalmoscope a clearly marked anaemia of the papilla of the optic nerve. But the beneficial effect of chloroform may perhaps be explained by assuming that it is an antidote to all poisons which produce spasms, and that here also a toxin must be held to represent the original cause of eclampsia (ptomato-tetanin). But these are merely hypotheses, and more exact etiological and pathological investigation is greatly to be desired.

#### CATALEPSY—TORPIDITY.

**Definition and Nature.**—Catalepsy is a peculiar periodic morbid condition, characterised by contraction of the body muscles, whilst passive movements and changes of posture can be made with remarkable ease, consciousness and sensation being at the same time lessened or even quite suspended. Some hold that this cataleptic rigidity arises from a transient heightening of the normal tonicity of the voluntary muscles, and others attribute it to an abnormally increased resistance of conduction within the motor ganglia, whereby the excitability of the muscles is reduced by the will and by external irritants. The disease occurs in our domestic animals both as an independent primary affection, and also secondarily in the course of other nervous diseases in the form of so-called cataleptic fits. But in general it seems to be extremely rare as an independent disease among our animals, and has hitherto only been noted in solitary cases, among dogs, horses, cattle and wolves. It is commoner in man, in whom it represents a kind of hysteria.

**Etiology.**—The causes of catalepsy are not yet known. Violent emotion is often blamed (terror), but this can only be regarded as contributory. The chief factor must be sought in an as yet not clearly understood nervous predisposition, for

the disease has many resemblances to the neurosis called hysteria in man.

**Pathological Aspect.**—Positive pathological changes of the central organs are as conspicuous by their absence in catalepsy as in eclampsia or tetanus. The disease must rather be regarded as a purely functional neurosis of the brain and spinal medulla. Occasionally certain secondary changes of the muscles are found which have some resemblance to those occurring in tetany, especially haemorrhage, opaque swelling and fatty degeneration of the muscles of the skeleton, waxy degeneration of the cardiac muscular fibres, and also haemorrhages in stomach and bowels (Fröhner).

**Symptoms.**—The attacks generally occur quite suddenly, but more rarely certain warning signs are noticed, such as restlessness, excitement, change of appetite. The rigidity begins in the muscles which happen to be in action, but spreads very rapidly over all those controlled by volition. As the animals are thus deprived of all power of voluntary movement, they stand motionless, like statues, and as though charmed, in the posture in which the fit surprised them. The affected muscles feel tense and firm, and occasionally stand out as if chiselled. At first they offer more or less resistance to any passive change of attitude; but this grows gradually fainter, and it is then possible to bring the limbs into all kinds of postures, in which they will remain unchanged for a long time (so-called wax-like flexibility, *flexibilitas cerea*). Consciousness and sensation are more or less considerably disturbed. Fröhner observed a perfect ecstasy in a cataleptic dog, a hypnotic state strongly reminding one of the descriptions of somnambulism in man. The eyes are rigid, appear dull as though veiled, and their pupils are at first enlarged and then contracted to the size of a pin-head. During the attacks the sensibility of the skin and mucous membranes is diminished or completely paralysed, and so, too, are the organs of sight, hearing and smell. The urine passed immediately afterwards contains albumen, and in dogs also much biliary pigment.

The course varies much in duration, and the attacks may recur. Spinola fixes their length at from a few minutes to an hour and longer; Hertwig speaks of them as lasting several weeks (?); Fröhner observed two attacks in a dog with a duration of 12 and 30 hours. The condition is not as a rule

fatal, but Hertwig says that animals may die of exhaustion brought on by interrupted consumption of food and drink. Fröhner saw one dog die on the seventh day, after the cataleptic rigidity had been followed by relaxation of all the muscles, including the involuntary muscles of the bowels and bladder, with a fall of bodily temperature and increasing coma.

**Therapeutics.**—Respecting the treatment but little is known. The narcotics which might perhaps be administered (morphia, bromide of potash) have been too little tested as to their operation, and are of doubtful value. Perhaps one might try to restore the disturbed reflex excitability by cold douches or the electric current.

#### CHOREA. ST. VITUS'S DANCE.

**Definition.**—Under the name of St. Vitus's dance, we understand in man continuous involuntary twitching movements of the body muscles, mostly irregular and seldom with any apparent purpose, which are complicated with more or less decided disturbances of the sensorium and of sensation. The disease has therefore been called muscular restlessness, muscular delirium or convulsions. The cerebrum has lately been assumed to be the seat of this neurosis, and capillary embolism of the corpus striatum and the thalamus opticus has been especially suspected as its cause, because chorea occurs in man with striking frequency along with acute articular rheumatism, endocarditis and valvular defects. The severance of the spinal cord in dogs suffering from chorea carried out so successfully by Chauveau and others seems to indicate that this organ also has something to do with the pathogenesis of the malady. How far the analogy between chorea in domestic animals and in man holds good is difficult to say with the small number of recorded cases before us and the manner in which many of them are described. But it seems as though simple cerebral and reflex spasms, perhaps even eclamptic and epileptic fits, had often been confused therewith. The spasms in dogs during distemper are not choreic, but epileptiform; but a symptomatic development of St. Vitus's dance is sometimes observed after recovery from distemper.

**Etiology.**—The causes of chorea are unknown. Young, feeble and anaemic animals suffer most frequently; probably

a nervous predisposition plays the chief part. The complaint is said to have been observed in horses, cattle, dogs, cats and pigs.

**Symptoms.**—From the reports of numerous observers it appears that the characteristic involuntary twitching movements are sometimes confined to the muscles of the head and neck (nodding, shaking the head), and again are spread over the entire muscular apparatus of the body (spasmodic motions, sometimes automatic or like a dancing puppet). Hering noticed in a horse continuous up and down movement of the head and of one fore leg; Leblanc, irregular twitchings over the whole body, whereby first one foot, then the head, the fore quarters, the hind quarters, an ear, a lip, an eyelid, etc., were moved. Anacker observed in a cow rhythmical movements of the head and neck from side to side, whereby the hind feet danced up and down as though walking. Schleg saw also in an ox continuous swaying to and fro of the head, the forelegs being thrown high up when walking. Hess and Verwey found clonic rhythmic spasms in young pigs over all the muscles of the trunk; some of them constantly flung their heads about, others kept making lifting movements with the rump, which was first raised into the air and then sunk to the earth. The *course* of St. Vitus's dance in our domestic animals appears to be chronic. Hess observed recovery in young pigs in from seven weeks to several months; and Leblanc noted a case in a horse lasting more than a year. The *prognosis* does not, however, seem to be unfavourable.

**Therapeutics.**—As in the other neuroses, the narcotics, chloral hydrate and bromide of potash are recommended. Legros and Onimus saw a choreic dog restored to quietness after a clyster of 50 grains of chloral hydrate. We can confirm these statements, but would remark that the sedative effect of narcotics is mostly only fleeting. Palombo says that he cured a dog by dipping it five or six times daily in cold water. Oxide of zinc and arsenic are also used. The latter we administer in the form of Fowler's solution, usually for the chorea which remains behind after distemper: Fowler's solution and cinnamon water of each equal parts, give from 5 to 10 minims daily.

## VERTIGO. DIZZINESS.

**Definition.**—Under the term of vertigo we describe periodical and suddenly recurring abnormal sensations respecting the relation of an animal to its surroundings, viz., an unusual feeling of movement, with a loss of the sense of equilibrium and consequent incapacity to remain erect. The nature of these morbid sensations is explained by functional or anatomical disturbance in the central organ for co-ordinated locomotion, i.e., in the cerebellum. It is true, however, that purely functional disturbances are less frequently the cause of vertigo than the multifarious anatomical changes. Vertigo is mostly only a symptom of other diseases and is but rarely a pure neurosis. It is well, nevertheless, to discuss it here separately, because it is considered in many lands, and we believe justly so, as a defect in warranty, and is therefore a question of great forensic importance. Viewed merely from the clinical standpoint it has only the value of a symptom, and should strictly be relegated to the realm of general pathology.

**Occurrence.**—Vertigo occurs most frequently in horses and dogs, more rarely in such animals as cattle, pigs or sheep. Full-blooded, well-nourished, and but little worked horses of medium to advanced age are most liable to attack. Spring seems to be among the seasons that in which vertigo is most rife; to which the prolonged winter sojourn in the stable, the rapid changes of temperature in spring, combined with the change of coat then in progress, all contribute. The condition is generally observed when the horse is in motion, more rarely in the stable, and usually only when being driven, much less often in riding. Some horses, indeed, which are habitually subject to vertigo in harness, are quite free from it when ridden. Many animals have regularly every year a few attacks of dizziness as winter passes away, and are free all the rest of the year. Among dogs the defect is noticed, especially in aged draught-dogs with heart-disease.

**Etiology.**—I. The causes of *independent essential vertigo* are, like those of all pure neuroses, unknown; there being no pathological changes in the cerebellum. In veterinary nomenclature this form has hitherto been known as "brain dizziness."

2. Those cases may be termed *sympathetic vertigo* which can be traced to permanent intra-cranial pathological changes. Among these belong tumours of the brain, of which cholesteatoma may be cited as a cause of the disease in horses, also encephalitic foci of softening, chronic inflammation of the cerebral membranes and affections of the cerebral vessels. Fugitive and non-periodic fits of dizziness are also observed in acute affections of the brain, e.g., in acute inflammation, or in acute anaemia brought on by severe loss of blood, or by rapid emptying of fluid accumulations from the body cavities, or of gas from the paunch.

3. The so-called "congestion vertigo," caused by *chronic disturbance of circulation in the brain*, is the form oftenest observed. Various diseases of the heart, vessels and lungs, which are accompanied by a congestive hyperæmia of the lungs, lie at the root of the condition. This congestive hyperæmia is in its effect precisely the same as an arterial anaemia of the brain, and excites the cerebellum partly by a lack of oxygen and partly by excess of carbonic acid. Thus we observe fits of dizziness in dogs in the course of valvular defects, in horses also in consequence of chronic, degenerative lung diseases, aneurysms of the pulmonary artery, etc. They are also very often caused by compression of the jugular veins in the neck by collar or breast harness, or by too-tight throat-straps or a too short bearing-rein (the latter particularly if combined with a tight snaffle). The operation of these causes is intensified by exhausting movement, great heat, warm and stuffy stables, full-bloodedness, etc. General chronic anaemia is also frequently accompanied by attacks of vertigo.

4. *Sympathetic* (consensual) or *reflex vertigo* arises, like reflex epilepsy, for which it is not seldom mistaken, through the transmission of a peripheral excitation to the cerebellum. The notoriously common "ocular dizziness" is included here. Among its causes are reckoned eye-blinkers with very shiny, smooth inner surfaces; blazing sunshine; strong reflected light from shining surfaces (lakes, rivers, pools, or snow when illuminated by the moon); rapid alternations of light and shadow in avenues of trees; as well as certain diseases of the eye, especially incipient amaurosis. Fits of vertigo are also observed after irritation of the ears, especially of the external passages, by foreign bodies; and in sheep during the disease caused by the larvae of *cœnurus cerebralis*. Whether intestinal worms can produce vertigo, as some affirm, is still uncertain.

So-called *abdominal vertigo* is presumably caused by certain diseases of the abdominal organs, especially by chronic gastric catarrh and intestinal catarrh, engorgements in the portal venous system, and by liver complaints (see Vol. II., p. 234). Cases in which horses manifest dizziness when turning a windlass or gin, or when being transported by rail or ship, must be regarded as *physiological vertigo*. In animals, as in man, true sea-sickness takes place, especially in dogs, being rarer in horses. *Toxic vertigo*, i.e., dizziness caused by poisonous food (*equisetum*, *solanin*, *lolium temulentum*, sedge-grass, alcohol) does not belong to this chapter really, and will merely be mentioned. The same applies also to the so-called *pasture-dizziness*, which is likewise certainly of toxic origin.

**Symptoms.**—The phenomena of vertigo are fairly simple. The horse suddenly shows a slower pace or stops entirely, makes shaking, nodding movements of the head, trembles, snorts, sways, staggers, straddles with its legs, presses backwards or sideways, holds its head up, or on one side, leans against the carriage-pole or against the next horse (if in stable, against the wall), turns a few times round in a circle and then hangs in its harness or in the halter. It betrays fear and uneasiness, sweats frequently and, in severer cases, cannot stand upright, but, if driven further, tumbles over, when it lies quiet for some minutes and then struggles to its feet and, after a brief interval of lassitude, appears quite well again. During the attack consciousness is lost, sensibility of skin and mucous membranes is reduced or quite gone, the pupils are dilated, pulse and breathing quickened and occasionally faeces and urine are involuntarily passed: dogs frequently vomit. The *duration* of the individual fits is very various, but is usually much shorter than in epilepsy, averaging from one to two minutes, in severer ones about five minutes.

The *differentiation* of vertigo from epilepsy is based mainly on the absence of spasms in the former. In horses apparent attacks of vertigo occur sometimes through pressure of the neck-piece of the bridle upon the region of the base of the ear (Wilhelm).

**Therapæutics.**—The treatment of vertigo should be guided entirely by its causes. These one must first of all endeavour to remove. Thus many horses, accustomed to stand too much in their stalls, have been cured by daily use and hard work. If the cause can be traced to a certain form of blinkers, they must be removed or altered. According to Baker, shading the eyes with a piece of leather 2 to 4 inches broad, passing in front

from one blinker to the other, has done good service. Straps which are too tight must be slackened, horses working a gin or other roundabout must be blind-folded. During an attack try to support the horse and cover up its eyes, if possible, take it out of the shafts, and see that everything be removed which might damage it in case of fall. If any cold water be at hand, the attack may be warded off or shortened by pouring it on the animal's head. Finally, as a prophylactic occasional derivation to the bowels by laxatives is to be commended, but no blood-letting. The vertigo accompanying heart-disease should be treated with digitalis.

**Menière's Disease.**—Peculiar fits of dizziness in man arising from disease of the semi-circular canals of the inner ear (*vertigo labyrinthina*) are known by this name. Fleming has observed similar cases in horses. They manifest themselves in the sudden shaking of one ear with sharp inclination of the head to that side, lateral turning movements, excited looks and tumbling over to the same side. But consciousness remains undisturbed. In a few minutes after falling the horses get up again, but often continue for some time to be much excited. We have ourselves seen similar cases in horses.

**Basedow's Disease.**—By this name we indicate in human medicine a neuropathic affection of the cervical sympathetic, which has not yet been etiologically quite cleared up, but which is characterised by three essential main symptoms which are genetically connected with each other.

1. Palpitation of the heart and vascular pulsation.
2. Swelling of the thyroid gland (*struma*).
3. Exophthalmus of both eyes (*goggle-eye disease*).

In many cases diseased changes in the lower cervical-ganglion of the sympathetic have been found as causes of the disease, and it is therefore supposed to arise from an inflamed condition of the sympathetic. Others suppose a central origin (*vaso-motor centre*). The complaint is frequently complicated with other brain diseases (*epilepsy, mental disease*). In recent veterinary literature five cases of Basedow's disease have been reported, viz., two each in horses and dogs and one in a cow. Jeswejenke observed in a four-year-old full-blooded mare, after a race, palpitation, accelerated pulse, growing weakness, swelling of the thyroid gland, and also, sixteen days later, excessive exophthalmus of both eyes, whereby the eyes were so strongly forced outwards that the eyelids could no longer cover them. The horse died after being ill for four weeks. He saw a similar case in a seven-year-old pug bitch, but she was cured after being treated for three months with iodine. Röder described the condition in a cow fourteen years ago. Here also severe exophthalmus was observed with strabismus convergens, palpitation, quickened pulse, and also a struma as large as the fist. The disease had then lasted four years. Cadot found in a horse hypertrophy of the heart and thyroid gland (there was no exophthalmus). An interesting case in a dog has been described by Albrecht.

## CHAPTER XIX.

### APPENDIX TO DISEASES OF THE NERVOUS SYSTEM.

#### CRIB-BITING.

**Definition.**—This may be regarded as a species of playfulness, or as a bad habit, and consists in involuntary gulping in of the air; either alone or mixed with saliva worked into a froth. So far back as 1664, Solleysel correctly recognised crib-biting as such, when he compared it to smoking and taking snuff in men. It occurs oftenest in horses, but is not rare among cattle; one case has been observed even in a pig. Among horses the animals kept for purposes of luxury, thoroughbred racehorses, stud and cavalry horses show the vice most frequently, though ordinary working horses do not escape it. The exact process in crib-biting is that, during a temporary interruption of breathing and visible contraction of the muscles lying on the anterior margin of the throat (sterno-thyroid, omo-hyoid, sterno-hyoid), the larynx and base of the tongue are drawn downwards, whereupon the upper part of the pharynx becomes filled with air, which, during the then following act of swallowing and return of the larynx and tongue-base to their former positions, partly escapes forwards and is partly swallowed, by which one or two so-called champing sounds are produced, i.e., peculiar tones like belching. Either all or a part of the air may escape forwards, or the whole may be swallowed. The older view that crib-biting was, on the contrary, an eructation of gases from the stomach, like the belching of cattle, and was therefore always a sign of digestive disorder or anatomical changes in the stomach, has now been pretty generally abandoned.

**Etiology.**—The causes of crib-biting vary.

1. The vice may develop *spontaneously* if the animals grow

wearied of long standing in the stall with nothing to do. Idleness is here the parent of vice. The animals endeavour to while away their time by licking the manger, crib, stalls, by biting all the wood-work within reach, by playing with their chain and its weights, etc.; and this more especially when tied up during the day. Other habits are also formed at the same time, such as sharpening the teeth, swallowing saliva and so forth. In particular the playing and striking with the lips, and sucking and swallowing air and saliva lead frequently to what we call crib-bitting. If horses are kept regularly at work, they never think of such things and have, in fact, no time for them. Thus Pansecchi relates concerning the horses of a regiment in which the vice was widely spread, that after they marched out to the war of 1866 only one horse retained the habit, but that on return to garrison it became as common as before.

2. In addition to its spontaneous appearance, it is certain that *imitation* has much to do with its spread; young horses in particular soon learn the vice from their neighbours, and it may thus pass through all the horses of a regiment, stud or stable. It spreads even among older animals by imitation, though not so rapidly, and a horse eighteen years old was observed to begin crib-bitting after standing for a time alongside a crib-biter.

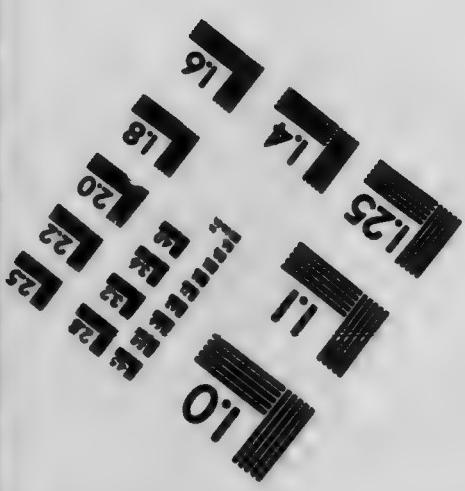
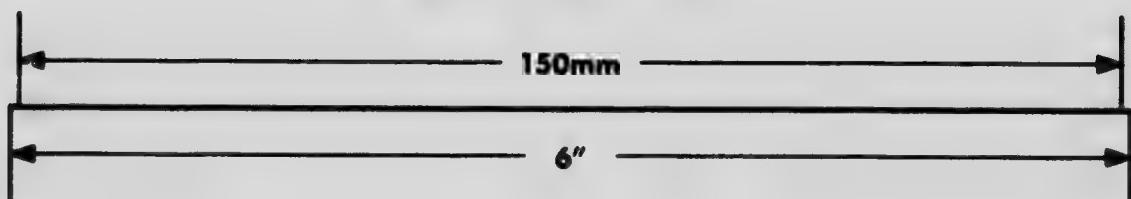
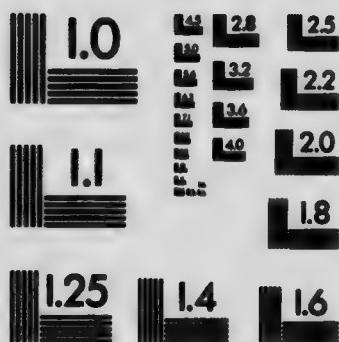
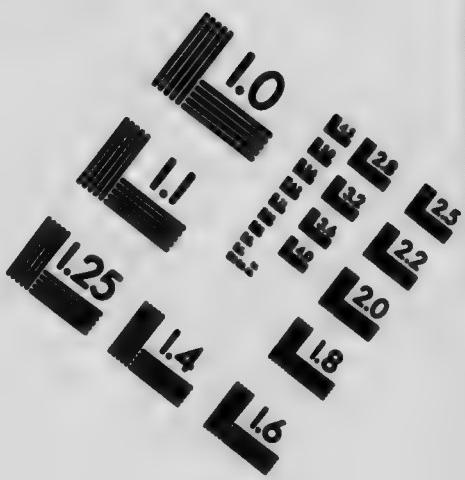
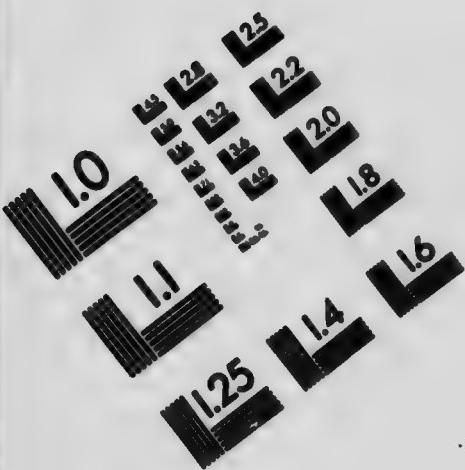
3. That the evil may be *inherited*, especially from the sire, has been fully demonstrated. Thus Collin reports that the 45 descendants of an Anglo-Norman stallion were all crib-biters, viz., one foal at 3 months old, two at 7 and 8, five at from 10 to 12 months, the majority in their second and third years, and one after 3 years.

Among other causes, too energetic grooming has been blamed, or unduly tightening the girth, both of which make the horse very restive, causing it to bite the crib with all its strength and to cling fast to it; also high feeding with but very small supply of water (training racers and hunters), which makes the animal try constantly to lick the manger, stall-sides, etc. (Sing). Hertwig's suggestion that the habit of crib-bitting is preceded by a feeling of discomfort in the bowels or a slight indigestion, has not much probability in its favour; for the chronic changes sometimes found in the stomachs of crib-biters *on post mortem* (dilatation of the stomach, thickening of its walls, chronic catarrh) are much more likely to be the results than the causes of the habit.

**Symptoms in Horses.**—There are several kinds of crib-bitting to be distinguished. 1. The commonest form is that of *crib-setting*, in which the incisor teeth, and sometimes those only of the upper jaw, sometimes those of the lower



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jaw, or even of both, are placed upon the edge or bottom of the manger. But this does not form the only support ; the bars of the hay-rack, halter, halter-chain, straps, carriage-pole, even the animal's own fore-leg, knee or hoof, are all used to rest the jaw upon. Günther saw foals use their mother's hock-joint for crib-biting. In rarer cases the chin or the top part of the throat is laid on the manger instead of the teeth. 2. *Crib-biters* seize with their incisor-teeth any object, such as the edge of the manger, a nail, etc. We saw a horse which laid hold of the wall of the stone barrier as far as it could open its mouth. The essential phenomena then in crib-setters and crib-biters are that the animals press against the point of support, open their mouths as for eating, bend back their necks, stretching tightly the muscles on the anterior edge of the neck so that they stand out in knobs, and then emit one or two belching sounds. Thereupon they let go the point of support, and the act is ended. 3. The so-called *air-snappers* or *wind-suckers* perform the action freely without resting the head on anything. They stand back from the crib, bend the head down upon the breast, make certain peculiar movements with the lips as in taking food, jerk the head and neck quickly upwards and snap air in with the mouth, when a loud tone is heard. Sometimes we see repeated snapping of air with an ever deeper sinking and forward movement of the head (like a swimming dog making several vain attempts to secure a floating stick).

The earliest signs are generally playful movements of the lips and tongue and especially an increasing desire to lick and to swallow saliva. Crib-biting is occasionally only done at certain times, and again with extraordinary frequency, and by some animals at every hour or even minute, indeed every four or five seconds. But various animals show great differences. Some only do it in their stalls or when they believe themselves unobserved, others during every unoccupied moment, others again only just before feeding, some immediately after, while some do it only while eating or chewing, and when swallowing their food very rapidly. The most serious cases are those of crib-biters which practise the vice until they are quite blown out with air. During sickness or while living under very unusual conditions, the practice is generally discontinued.

The disadvantages of crib-biting are first, that much fodder is wasted, and that consumed is but poorly made use of, and secondly that neighbours are disturbed and may be contaminated by evil example. True signs of illness are very often absent.

But in other cases crib-biting is the cause of chronic disorders of digestion and nutrition, of tympanitic inflation of stomach and bowels, and of colic; on these grounds the habit has an essential pathological connection with digestion and its entire apparatus. As further consequences of crib-biting one has observed (Hell, Hartenstein and others) in animals which had brought the vice to a certain point of perfection an invariable hypertrophy of the sterno-maxillaris muscles and of the omo-hyoid muscles (caused by over-exercise).

**Symptoms in Cattle.**—The habit occurs here, too, in variously modified forms, but shows itself most frequently as follows:—The animal opens its mouth widely, raises the head, stretches out its tongue and persistently swings it to right and left, thereby beating its saliva into foam, which collects in the corners of its mouth and not unfrequently smears its outer cheeks; finally, with a clucking noise this is swallowed along with air. By this process, oft-repeated, the paunch becomes exceedingly and tympanitically inflated, when the practice is suspended and the air gradually ejected by repeated belchings.

That crib-biting may assume a different form in cattle is proved by a case reported by Weinmann, in which an ox stood still with close-shut mouth and constantly sucked in air through the corners with a peculiar wheezing sound, whereby its paunch became inflated like a drum, when the sucking ceased, and the air gradually came back by eructation. Johne also observed a cow which jammed her head firmly into the corner of the food-trough and by means of her tongue, and to the accompaniment of a singular clucking noise, pumped herself full of air, when she stood quite still for thirty or forty seconds, and then audibly ejected the air in an unbroken stream and with evident signs of satisfaction.

In the Dorpat hospital crib-biting was observed among *pigs*, which placed their incisor-teeth, just like horses, on the edge of their food-trough and emitted a similar sound.

**Diagnosis.**—A just recognition of crib-biting is of especial forensic importance, because in some countries it is one of those defects against which warranty can be legally demanded (in Bavaria, Württemberg, Baden and Hesse the period of warranty is eight days). Diagnosis is mainly based on proof of the muscular contraction on the anterior border of the neck and on the characteristic crib-biting sound. If air be swallowed, we may

demonstrate the fact, if familiar with the animal, by auscultation or by compression of the pharynx with the hand on two different spots, as in the latter case a downward gliding, wave-like movement is felt. A wearing-down of the teeth may also occur in animals which are not crib-biters. Confusion with other forms of playfulness in which air is inhaled must be avoided; such are simple biting of the crib, sharpening the teeth thereon, saliva-swallowing, blowing with the lips (trum-peting), licking, striking with the mouth, etc.

**Therapeutics.**—As a prophylactic, isolation of the affected animals should be first attended to. Complete cure is possible at the beginning of the evil, but inveterate crib-biters are incurable. The following remedies have been tried:—

1. Intimidation by punishment; result doubtful.
2. Exhausting labour, avoidance of prolonged inactivity; result good.
3. Removal of all objects on which the jaws, etc., can be laid, tying up the animal, reversing it in the stall; result generally not very enduring.
4. Placing the manger on the ground; use of a nosebag; result doubtful.
5. Introduction of movable mangers, covering them with sheet-iron, fixing spikes on the edges and bottom of the trough; result doubtful.
6. A closely-fitting muzzle; result at first good.
7. Various head-straps, either in the form of a simple, narrow leather band, drawn tightly round the neck in the region of the larynx (which, however, compresses the blood-vessels too straitly), or broader straps with metal tongue, elastic steel clasp and sharp spikes (Ringheim and Burdajewitz's head-straps). But the latter may produce phlegmonous swelling of the neck as the result of injury inflicted.
8. Günther's crib-biters' pipes, which are hollow, perforated metal pipes inserted as mouth-pieces. Also various crib-biters' halters.
9. Severance of the omo-hyoid (Gerlach) or sterno-maxillaris muscles (Hertwig, Hell); but, according to the reports of Hering, Bassi and others, the result here also is not durable.

#### JIBBING.

**Definition.**—Jibbing is a vice of horses consisting in persistent, obstinate and conscious refractoriness against the require-

ment of an ordinary duty, and it not only greatly reduces the animal's usefulness but constitutes a general danger. We may distinguish between absolute and relative jibbing. The former means that the animal is useless, for all purposes, for driving as well as for riding. The latter, which is much commoner, is disobedience only in certain kinds of work. There is a rider's jibbing and a carriage-jibbing, a jibbing in single harness and a jibbing only in double harness, as saddle-horse or as led horse. The evil is commonest in mares, also in Polish horses and half-wild breeds; it is less frequent in those from marsh-lands. Light bays have an especially bad reputation.

**Etiology.**—The cause of jibbing is usually to be found in improper or brutal treatment of the animal when being broken in and trained to ride or drive, or in some senseless and cruel punishment. To these influences high-spirited and well-bred animals are very susceptible. But malicious teasing and irritation, as well as recovery from painful skin-injuries may give rise to jibbing. Among temporary causes ruttishness in mares must be named. How far heredity or pathological conditions of the brain may conduce to jibbing is not with certainty known.

**Symptoms.**—The phenomena of jibbing are partly active and partly passive, but both are frequently combined.

1. In *active jibbing* the animal stands still when at work and offers active resistance to all attempts to make it go forward. It skips here and there, presses to one side, throws itself on its fellow horse, rears up, leaps over the traces, kicks out with the hind feet, so that frequently harness, pole, and the front of carriage or cart are broken, even flings itself on the ground and strikes out with all four feet. Saddle-horses try to throw their riders, rearing high up and bucking, or seek to knock him off against trees, walls, etc. Not unfrequently they fall quite over in their attempts. At such times horses manifest great excitement, a fiery eye, palpitation, reddened mucous membranes, trembling, quickened breathing and sweatings. In rarer cases this refractoriness increases to perfect mania, so that at last the animals are led away in a half-stupid condition. Great exhaustion generally follows on such attacks. Such animals usually appear quite good-tempered when in the stable.

2. In *passive jibbing* the animals while at work suddenly stand as though rooted and spellbound, back into the harness

and stop the carriage intentionally and cannot by any manner of means, kind or severe, be got to move a step forwards. Mostly they attempt to turn back. If left to themselves they usually go on of their own accord after a certain time. In other cases the driver must get down and unharness the animal, or else return home.

These two forms of jibbing may be variously combined. Very often the animal will obey if its load be lightened, or on the way back to the stable ; but if heavily laden or in going up-hill, etc., will continue refractory.

**Diagnosis.**—As jibbing counts for one of the chief defects under warranty in some lands (the period being in Prussia 4 days, Saxony 5, Hesse 9 and Austria 30 days), one must consider the possibility of mistaking it for other conditions. It must be clearly stated that by jibbing is understood a conscious refusal to render obedience in a service corresponding well with the animal's conditions. Jibbing is distinguished from sleepy staggers by the consciousness and deliberateness of the act. Then a horse must not be regarded as jibbing if it refuse to work when put to unaccustomed or to excessive toil, or because of bodily pain (uncomfortable collar or saddle, etc.) or illness. The average power of traction of a horse of medium strength upon a moderately good road is from 30 to 40 cwts. ; on a bad road about a ton, on a well-paved one about 50 cwt. Finally, jibbing must not be confused with shying.

**Therapention.**—Confirmed jibbers are incurable. Only in slighter cases, that is, during the development of the vice, can we obtain improvement, and this solely by psychic influences, by goodness and kind words, above all, by patience, but never by compulsion. In general, jibbing is regarded as incurable, although it may occur that jibbers appear to lay aside the vice for a while under well-accustomed surroundings.

#### NERVOUS DISEASES OF POULTRY.

1. **Hypersomnia of the Brain and Haemorrhage of the Brain** (apoplexy) occur among male birds, especially at the breeding season, after mechanical injury, as well as among young poultry if exposed to burning sunshine. Friedberger also noticed them in the course of chicken-diphtheria. The symptoms are dizziness, staggering, stupor, dilated pupils, rotary movements, motion backwards, irregular action of wings and feet, as well as peculiar epileptiform spasms, which are often caused by a mere

touch. The head and neck are often so twisted that when the bird is standing its skull is turned to the ground and the beak directed forwards. The creatures collapse, make straddling movements of the legs and twitch their wings, so that the prostrate birds are turned round on the ground. They then stand up again, unless the attack end apoplectically. As a *therapeutic*, cold is recommended and also purgative (for fowls a tablespoonful of castor-oil, or  $1\frac{1}{2}$  grains of calomel, or 8 grains of jalap root). (Zürn).

2. **Epilepsy** seems not to be at all rare in poultry. We have seen several cases in the different kinds of birds. In a case observed by Friedberger a goldfinch made flapping movements with outspread wings on the floor of its cage, had violent clonic spasms of the muscles of its extremities, fell on its back, or remained sitting on one spot with straddled legs, its head being spasmodically jerked up and backwards, its bill and eyelids opened and shut and its eyeball rolled, whereupon consciousness quickly returned. Bromide of potassium is to be used here, as for other animals (a 1 per cent. solution as drinking water).

## CHAPTER XX.

### DISEASES OF THE RESPIRATORY SYSTEM.

#### I. DISEASES OF THE NASAL CAVITY.

##### ACUTE NASAL CATARRH OF HORSES. CORYZA.

**Etiology.**—The causes of acute nasal catarrh, or superficial inflammation of the mucous membranes of the nasal cavity are various.

1. *Primary*, independent, acute nasal catarrh is most frequently produced by cold, especially in spring and autumn. Limp, pampered and youthful animals are particularly liable to it. The much greater frequency of acute nasal catarrh in horses than in other animals, such as cattle, may be explained partly by their greater exposure to cold, and partly by the greater width of their nostrils, whereby the mucous membranes are more exposed to external irritants. Among the latter we may mention, the inhalation of dust from the streets or from fodder, of smoke, fungi, spores, etc., which may be floating in the air after distribution of food, also from the ammoniacal air of close and crowded stables. The possibility of infectious influences in the production of coryza cannot be denied, as its occasional epizootic appearance points clearly to some such cause. But nothing positive is as yet known on this head.

2. Acute nasal catarrh occurs *secondarily* with very great frequency as an accompaniment of other diseases, especially of infectious diseases and diseases of the breathing organs. Not seldom the catarrhal process extends from a primarily affected mucous membrane, as, e.g., from the pharynx to the nasal cavity.

**Symptoms.**—The first signs of acute nasal catarrh consist in a diffuse hyperæmia (less frequently petechial), as well as in dryness of the nasal mucous membranes. The expired air

is somewhat hotter, owing to the membrane being more fully charged with blood (stage of inflammatory irritation). A few days later the chief symptom of the disease appears, viz., discharge from the nose; the mucous membrane is now shiny, swollen and often quite oedematosly saturated (stage of secretion). The nasal discharge, which is usually bilateral, is at first serous, as clear as water, and of very thin, watery consistence, and in slighter cases this appearance does not change. But if the catarrhal process go forward, the nasal outflow, owing to increased mucous secretion, becomes glairy, slimy, and also opaque and turbid, this arising from simultaneous active desquamation of epithelium. Lastly, the secretion becomes pus-like (serous, mucous, purulent catarrh) owing to the admixture of numerous white blood-corpuscles. Horses often snort, and the vicinity of the nostrils is usually found encrusted with dry discharges of mucous and pus-like character. Occasionally the lymphatic-glands of the throat are found slightly swollen.

The duration of acute nasal catarrh averages eight to fourteen days. Recovery usually follows without any assistance, the discharge gradually abating. But the disease may from the outset be accompanied by severer signs, such as shivering fits, loss of appetite, slight rise of temperature and of pulsation, and may spread to neighbouring mucous membranes, such as the frontal sinus, or, by way of the lachrymal ducts, to the eyes. Then great heaviness is observed in the head, catarrhal conjunctivitis, weeping of the eyes and avoidance of the light; very often this condition is accompanied by laryngitis, stomatitis, catarrh of the superior maxillary sinus, etc. Finally, the condition may become chronic.

**Therapeutics.**—Treatment is only necessary in more pronounced cases; lighter ones recover just as quickly of themselves. The most effectual means is the local treatment of the nasal mucous membranes by inhalation of steam and hot vapour, to which one may add a little creolin or carbolic acid. It is hardly necessary in acute catarrh to rinse the nasal cavities with astringent fluids. Internally anti-catarrhal salts (neutral salts, common salt) may be administered in combination with aromatic vegetable powders. More important is good ventilation of the stable and bringing the animal out of doors, if the weather be favourable; moreover, a supply of green fodder, turnips, etc., or free grazing in the open, are all commended.

## CHRONIC NASAL CATARRH OF HORSES.

**Etiology.**—In contrast to the acute form just described, chronic, primary nasal catarrh is much rarer among horses ; it is developed from the acute by greater intensity and diffusion and by longer and repeated operation of the causes. More common and much more important clinically is the secondary nasal catarrh caused by other morbid processes. Of such we must especially mention : 1. Glanders, which often exhibits merely a chronic nasal catarrh. For this reason chronic nasal catarrh is always a very suspicious sign in horses, which requires watching with great care. 2. Catarrh of the nasal accessory cavities (superior maxillary sinus, frontal sinus) often reveals itself only by a chronic nasal discharge. 3. Tumours, parasites, abscesses, etc., in the nasal cavity. 4. Diseases of the teeth, especially suppurative periostitis of the alveoli of the teeth, with their complications. 5. Chronic affections of the breathing apparatus in general, as well as chronic constitutional diseases, such as leucæmia and anaemia.

**Symptoms.**—During chronic nasal catarrh the mucous membranes of the nose are pale or cyanotic and, if prolonged, they become thickened and are traversed by dilated veins. The nasal discharge is sometimes glairy, slimy and like the spawn of frogs, or it is putrescent, yellowish or dirty-grey in colour, smearing the nostrils, and occasionally smelling badly. Frequently one may perceive a stripe upon the skin beneath the nasal opening and corresponding to the point at which the catarrhal secretion is discharged, upon which the skin-pigment is lacking. The quantity of the discharge varies much, it may grow to a veritable stream of mucus (blennorrhœa). Sometimes it ceases entirely for a brief period ; and again a very great quantity may be discharged suddenly, as after movement, or on lowering the head. When the flow is from both nostrils, this generally indicates a primary catarrh ; when from one only, a secondary (glanders, new growths in the nasal cavity, dental trouble, etc.) ; with the latter we often find also swelling and induration of the sub-maxillary glands.

As a peculiar complication of chronic nasal catarrh, when much prolonged, we sometimes find on the nasal septum, so-called erosion-ulcers which are flat, superficial losses of substance, from a pin-head to a lentil-seed in size and with sharp,

unthickened edges. These are sufficiently distinguishable from the ulcers of glanders by this description. Nevertheless, we must state that such erosion-ulcers occur also in glanders, as we have had occasion several times to observe, because the latter gives rise secondarily to chronic nasal catarrh; their presence therefore affords no presumption against the existence of glanders. The erosion-ulcers (formerly known as catarrhal ulcers) heal without leaving any scab or scar, by which also they may be known from true glandulous ulcers.

The duration of chronic nasal catarrh may extend to weeks, months, and even years, and the malady is in general difficult to cure. When much prolonged, hyperplastic processes may develop upon the nasal mucous membrane.

**Diagnosis.**—It is always very difficult, and sometimes impossible, to decide the question, so important for prognosis, whether we are dealing with a primary or a secondary chronic nasal catarrh. The best guide is often afforded by the course taken by the disease, which in a primary catarrh is more favourable than in a secondary. The fact also that the discharge is from both sides may help to decide in favour of the primary form. But in any case, careful and frequent examination of the nasal cavity with a rhinoscope is indispensable. For a veterinary hospital we recommend the special rhinoscope of Polansky and Schindelka, which renders it possible to examine fully the entire nasal mucous membrane of a horse. In many cases it is necessary to trephine the cavities of the nose, the frontal or superior maxillary sinuses, or experimentally to inoculate other animals with the catarrhal secretion, and this particularly if there be any suspicion of glanders. Compare on this point the paragraph on the differential diagnosis of glanders.

**Therapeutics.**—In this case also the treatment is essentially local, and consists either in the application of vapours of water, creolin, lysol, carbol, creosote, tar and oil of turpentine, which may be used one after the other in the order given, applying for a time steam only, and then the stronger vapour of carbol, etc.; or else in washing the nasal mucous membrane with astringent liquids (1 to 2 per cent. solution of sulphate of zinc or creolin, or a  $\frac{1}{2}$  per cent. solution of nitrate of silver, a one-per-thousand solution of sublimate, etc.). These are either sprayed in through the nostrils, or introduced into the

nasal cavity from above after trephining (Dieckerhoff). It is also of great service to rinse the nasal mucous membrane by means of a stout india-rubber tube or elastic stomach sound introduced through one of the posterior nasal openings. The value of internal remedies (ammonia, spirits of turpentine) is here very doubtful. But from a hygienic stand-point it is well to provide as fresh an atmosphere as possible, frequent sojourn in the open, cleanliness in the nostrils, manger, etc., with good fodder and attention to the skin. Finally, it is urgently advised to separate all horses suffering from chronic nasal catarrh as a precaution. Any treatment of the erosion-ulcers with drugs is to be avoided, as they are thereby hindered in their development and considerably altered, so that the most important guide in diagnosis of glanders is removed.

#### NASAL CATARRH IN SHEEP.

**General Remarks.**—The nasal catarrh of sheep has in so far an especial importance as that hitherto quite different diseases have been included under this name or its synonyms, "sheep-glanders" and "benign" and "malignant catarrhal fever." The last of these should be regarded as an infectious disease, in which the affection of the nasal membranes represents only a symptom. We think, therefore, that the following distinction should be made.

1. *Simple acute nasal catarrh* is in sheep of so slight a character that in most text-books it is not even mentioned. It occurs very commonly, chiefly in spring after prolonged sojourn in the stable, also in autumn from cold, and likewise after shearing, or from inhaling dust, etc. It shows itself in nasal discharge, sneezing and a wheezing respiration.

2. *Simple chronic nasal catarrh* represents the "benign catarrhal fever" of veterinary authors, and develops from the acute more readily when the producing causes operate for long periods (persistent windy, wet and cold weather, etc.). To young and feeble animals this simple chronic nasal catarrh may, if long continued, become even dangerous to life. It is manifested by a chronic muco-purulent nasal discharge, with blocking of the nostrils and formation of crusts, and is in some flocks very widely spread. (It may possibly be confused with pulmonary worm-disease.) The treatment consists in inhalation of tar-vapour, etc., and in administration of saline remedies.

3. "*Malignant catarrhal fever*," or "sheep-glanders," is by

far the most important of all the diseases classified as nasal catarrh, but obviously represents an infective malady. We do not therefore treat it here, but in the chapter on infectious diseases in our first volume, p. 54.

## NASAL CATARRH OF CATTLE, PIGS, DOGS AND CATS.

**General Notes.**—Primary nasal catarrh is not very common with other domestic animals, except dogs, nor of much importance. More frequent is the secondary form, which is an accompaniment of very different infectious diseases (distemper, pentastomum *tænioides*, verminous bronchitis, tuberculosis, malignant catarrhal fever), and has there been discussed. Concerning idiopathic, primary nasal catarrh in cattle and pigs nothing certain is known. But in dogs it occurs very often and is shown in running at the nose, sneezing and sniffling, rattling sounds both on inspiration and expiration. Sometimes the catarrh is purulent and haemorrhagic. Its cause is probably to be sought in cold; but a distinction between a simple and specific (as in distemper) catarrh is not always easy to draw. Treatment is seldom necessary. When desired, or when the catarrh is chronic, it must consist in inhalation of solutions of salts (common salt, ammonia) or of creolin-water ( $\frac{1}{2}$  to 1 per cent.) and also in internal administration of laxative saline remedies (common salt, Glauber salt, ammonia). Injections through the nostrils or other irrigations of the mucous membranes are attended with much difficulty in dogs, owing to the animals' resistance and the smallness of the nasal passages.

With cats a chronic, purulent, nasal catarrh sometimes occurs, with swelling of the nasal bones and abnormal thickening of the skin on the ridge of the nose (elephantiasis, rhinosclerom), during which, as in glanders and tuberculosis, the adjacent lymph-glands also become swollen. This usually incurable disease has great likeness to the somewhat common rhinitis gregarinosa of rabbits. The latter forms an accompaniment of coccidiosis described in Chapter VIII., Vol. II., and presents the aspect of an epidemic, malignant cold, with participation of the tarsal conjunctiva. Respecting the infectious rhinitis of pigs, known as "snuffing sickness," see Chapter I., Vol. I.

**SEVERE INFLAMMATORY CONDITIONS OF THE NASAL MUCOUS MEMBRANE (RHINITIS).**

(*Phlyctænular, croupous, follicular and diphtheritic inflammation.*)

**General Remarks.**—Severe inflammatory conditions of the nasal mucous membrane are caused in the first place by intensely active irritants, especially of infective character; they occur, therefore, mainly as signs of infectious disease. Their differentiation as phlyctænular, croupous, follicular or diphtheritic is consequently not the most important concern, as these pathological changes do not cover a clinical unity, but rather the same condition may be produced by several causes and *vice versa*. For instance, a croupous rhinitis may be just as easily caused by mechanical or thermal influences as by the contagium of strangles, and, on the other hand, strangles may have as a sequel either a phlyctænular, a croupous or a follicular rhinitis. Nevertheless, it is advisable to adhere to the above purely pathological definitions upon practical grounds, as the several inflammatory causes, and especially the infectious diseases at the root of these pathological changes, are not yet sufficiently well known.

**1. Phlyctænular Inflammation of the Nasal Mucous Membrane** occurs most frequently as a co-symptom of strangles (as so-called phylctænular strangles), also in very severe, simple acute nasal catarrh. It is nothing but a vesicular catarrh, in which small blisters rise upon the nasal membrane, at first clear like water, but whose contents may later become purulent. By the bursting of these blisters and drying up of their contents, thin scabs are formed, which fall off when the tissue is completely renewed.

**2. Croupous Inflammation of the Nasal Mucous Membrane** arises first from violent irritation of the membrane, as for instance, from inhaling smoke and hot air on an outbreak of fire. Strangles also may be accompanied by croupy rhinitis, but this is rare. Further, nasal croup may occur among horses, especially cavalry remounts, and sometimes enzootically, which gives much probability to the assumption of an infective cause. This form of croupous rhinitis must therefore be regarded as an infectious disease. Röll and Buck-

müller, who observed the disease pretty frequently in Vienna, describe its symptoms as consisting in a deposit upon the nasal mucous membrane of grey or reddish yellow, skin-like, croupy membrane, which is sometimes of considerable extent, and by suppuration is gradually pushed off. The subjacent mucous membrane is extremely hyperæmic, and, after the croupy membrane has been cast off, appears excoriated and bleeds readily. At the same time there is a viscid, yellowish nasal discharge, and, in severer cases, difficulty of breathing, fever, sensitive swelling of the lymphatic vessels of the upper lip, cheeks and lower jaw, as well as of the glands in the cavity of the latter. *Prognosis* is usually favourable, and *treatment* the same as for simple nasal catarrh. Owing to its infectious character, sick animals should be isolated. A similar infectious nasal croup has been occasionally seen in oxen. In contrast to malignant catarrhal fever, it is very benign and never complicated with affection of the eyes. It affects several inmates of the same stable at one time with high fever, their breathing becoming very noisy, but in about eight days they have mostly recovered. Berndt saw this singular infective malady in 16 cows of one byre.

3. The so-called **Follicular Inflammation** is an intense inflammation of the mucous glands of the mucous membrane and of the sebaceous glands in the skin round about the nostrils, during which the orifices are blocked and cellular and fibrinous products of inflammation accumulate, so that nodules appear upon both membrane and skin, which afterwards ulcerate. It cannot be a true lymph-follicular inflammation, such as occurs in swelling and suppuration of the intestinal follicles, because there are no lymph-follicles either in the nasal mucous membrane or in the skin; so that the name given above is obviously misleading. The affection seems to differ only from croupous rhinitis in its location, and not infrequently they are found side by side. Its causes must certainly be sought in the action of some infectious substance. It is certain that some of the cases cited are identical with a special form of strangles, and the rest appear to be caused by an infectious disease which at least bears a close resemblance to strangles. It occurs frequently in epizoötic form, and particularly in military or well-filled stables; but we have also met with it in carriers' stables. Its contagiousness is undoubted, and among cavalry we have seen it pass directly from horse

to horse. It begins with violent, acute nasal catarrh, during which the mucous membrane of the septum displays quite characteristic changes. In consequence of the swelling of the mucous glands its surface appears rough and as though covered by small granules. The swollen glands grow into yellowish nodules, which break on their upper surface, and are covered and surrounded by a membrane-like deposit, which can only be removed with difficulty or not at all. When it falls off, little flat, shallow ulcers, whose edges are very red and which lie close together in great numbers, are found in place of the nodules. These ulcers quickly heal without leaving any scar, their base being covered with epithelium growing from the edges. Nodules and ulcers of similar description appear also on the borders of the nose, around the nostrils, and also on the skin of the upper lip and lower edge of the masseter. From these ulcers inflammatory strands of the lymph-vessels often proceed to the much swollen lymph-glands of the cavity of the lower jaw, even to those of the neck and shoulder or anterior breast. We have observed swellings as thick as an arm, and corresponding to the course of the larger superficial lymph-vessels, along the mastoido humeralis muscle to the entrance of the breast. These swellings of the large lymph-vessels of the neck are hard and tense, like those on the head, and not seldom form abscesses. We also noticed in many cases conjunctivitis and even decided blennorrhœa. The *course* of the malady is mostly benign, and complete recovery usually follows in two to four weeks. Any confusion with glanders is scarcely possible with careful examination. The superficial and always numerous ulcers, which readily heal and leave no scar behind; the frequent spread of the same upon the outer skin; the presence of inflammation in the lymph-vessels; and the benign course of the disease all assure diagnosis. An examination of the oral cavities will guard against the not infrequent confusion with stomatitis contagiosa pustulosa; these cavities are almost always attacked in the latter disease. *Treatment* is the same as for croupous rhinitis (inhalation of vapour of water, creolin and carbol). It is well also to rub the swollen lymph glands and vessels with an ointment of iodoform, creolin, camphor, mercury or carbol. Direct treatment of the ulcers by touching them with nitrate of silver we consider unnecessary, and under some circumstances as rendering a differentiation from glanders more difficult;

**4. Diphtheritic Inflammation of the Nasal Mucous Membrane** has nothing in common with human diphtheria; it is rather to be regarded as merely a necrotic inflammation of the mucous membrane, which is extraordinarily rare as a primary disease, and is noticed, for instance, after the action of various very violent irritants which practically destroy the membrane. It is seen more frequently as a secondary accompaniment of several infectious diseases, as in acute glanders, the petechial fever of horses and malignant catarrhal fever of cattle, etc. See the chapter on Diphtheria, Vol. I., pp. 220 to 238.

*NEW GROWTHS IN THE NASAL CAVITY.*

**General Notes.**—Apart from the new growths occurring in glanders, ulcers and abscesses of various kinds appear in the nasal cavity, especially on the septum, in the fossæ or on the ethmoid bone, all of which cause at first a chronic nasal catarrh. As most of these occur only on one side, not seldom proceed to ulceration and are associated with swelling of the lymph-glands in the cavity of the lower jaw, they possess much clinical interest in horses, since they are very easily mistaken for glanders. For this reason they shall be here briefly discussed, although really belonging to the realm of surgery and only to be treated by operation. For further information see the differential diagnosis of glanders (Vol. I., pp. 282 to 286). The rhinoscope of Polansky and Schindelka is important for the diagnostic demonstration of these and other new growths in the nasal cavities of the horse.

**I. Fibromata** occur either as localised connective-tissue tumours, or as so-called polypi, either pedunculated or upon a broad base, or else they form flat, chronic proliferations of mucous membrane. Polypous fibromata are sometimes visible even from without, they keep up a chronic nasal catarrh and cause a wheezing, snoring sound by narrowing the affected nasal passage. The breath can often only pass through one nostril, so that, on placing the hand in front of the diseased nostril, no air-current, or only a very faint one, can be perceived, and by closing the sound nostril dyspnoea is at once caused. Occasionally we may see superficial necrosis and an ichorous nasal discharge with swelling of the glands upon one side. As the polypi grow, they may proliferate into the cavities of the

mouth and pharynx, or press the septum to the other side. The flat proliferations of mucous membrane produce a lumpy surface upon it and, like the polypous form, are followed by chronic catarrh, stenosis of the nasal passages, difficulty of breathing, and sometimes ulcerative decomposition with an unpleasant nasal discharge.

2. **Sarcomata** and **Carcinomata** are rare. They usually displace the neighbouring bones (of the nose, maxilla and palate), and may even up-lift the facial bones, forcing themselves into the oral and pharyngeal cavities, where they cause difficulty of eating and swallowing. They also often become ichorous, so that a foul-smelling, discoloured discharge from the nose is caused, with swelling of the submaxillary glands. Carcinomata also produce bleeding at the nose. The starting-point of sarcomata is usually the periosteum of the adjoining bones (osteosarcoma). Metastatic formations in the submaxillary lymph-glands are sometimes observed along with carcinomata and sarcomata.

3. **Angiomata** of the nasal septum arise from dilatation and new growth of vessels (angioma simplex), and frequently assume a cavernous character (angioma cavernosum). They are never sharply delimited, but extend superficially; their colour being dark brown or bluish-red. After mechanical irritation or increased blood-pressure caused by exertion, they easily bleed, so that bleeding at the nose results; they show also a great tendency to ulceration. The ulcers are often covered with blood-coagulae and have a reddish-brown base. When they heal, scars remain, as from glanders ulcers. Moreover, on ulceration of an angioma, a bloody or discoloured nasal discharge begins, with swelling in the submaxillary glands. As will be seen from this description, angiromata may be only too easily confused with glanders; and in many cases diagnosis is assured only by microscopical examination of the new growths.

4. As rarer forms of new growth we would mention: lipomata, rhinoscleromata, myxo-fibromata, epitheliomata, enchondromata, odontomata, osteomata, adenomata, actinomycomata, tuberculous and amyloid new growths, dermoid cysts, colloid cysts, ulcers which contain *Strongylus armatus*, and also, in dogs, the occurrence of hair upon the mucous membrane of the nose.

As further morbid affections of the nasal cavity we may also cite:

**abnormal enlargement of the concha and abscesses.**—The former is partly congenital and partly caused by chronic nasal catarrh, previous strangles, or by a creeping osteitis originating in the alveoli of the teeth. It sometimes very seriously impedes breathing and is usually complicated with chronic nasal catarrh. Abscesses in the nasal septum and in the cells of the ethmoid bone arise partly from wounds and partly in the course of catarrhal inflammation, and lead to destruction of cartilage and bone. They may even give rise to suppurative nasal discharge, bleeding from the nose and glandular swelling.

#### BLEEDING FROM THE NOSE. EPISTAXIS.

**Etiology.**—Bleeding at the nose is only a symptom, and consequently just as little an independent disease as haematuria. It occurs in all animals, but is commonest in the horse. Its causes may be of the most varied nature, and during life very often cannot be determined with certainty. The most important affections of the nasal mucous membranes are :

1. *Traumatic influences*, injuries, contusions.
2. *Abnormal blood-pressure* after severe exertion, e.g., after forced racing, in congestion of the brain, heart-defects, lung affections, etc.
3. *Vascular diseases*, especially angioma and varicose dilatation of the veins.
4. *New growths and ulcers*.
5. *Intense inflammation of the mucous membrane*.
6. *General disease with haemorrhagic diathesis* (strangles, haemophilia, petechial fever, anaemia, leucæmia, etc.). In horses a slight periodical nose-bleeding is often a symptom of glanders. Such bleeding in horses is therefore always of importance.
7. Respecting bleeding from the nose in dogs, compare the chapter on dochmiasis (Vol. II., p. 227).

**Symptoms.**—In epistaxis the discharge is usually only from one nostril, seldom from both; sometimes it is only in drops, at others in a slender stream. Not unfrequently the usual nasal discharge is merely streaked with blood. The bleeding is often only temporary, or it may be repeated several times; but in other cases it continues till the animal bleeds to death. In contrast to hemorrhage from the lungs, the blood is not frothy, and there is no cough.

**Therapeutics.**—When the bleeding is slight, no treatment is required. In severer cases we try first of all injections

of styptic solutions into the nasal cavity, especially a 5 to 10 per cent. solution of per-chloride of iron. If the bleeding be dangerous, the affected nostril must be plugged with cotton-wool soaked in creolin, carbol or sublimate, for which purpose, under some circumstances, the nasal cavity must be trephined. Styptic remedies may at the same time be internally administered: sugar of lead, tannin, sulphate of iron, ergot or hydrastis.

## CHAPTER XXI.

### DISEASES OF THE ACCESSORY CAVITIES OF THE NOSE.

*Inflammation of the Maxillary and Frontal Sinuses of Horses.*

**Occurrence.**—Among the accessory cavities of the nose the superior maxillary sinus is most frequently attacked by catarrh, particularly its upper and larger division, more rarely the lower part and the frontal sinus, and still more rarely the ethmoid cells and sphenoidal cavities. It is usually unilateral.

**Etiology.**—Catarrh of the sinuses may be first caused by traumatic influences (fracture or infraction of the bone), perhaps also by cold, but much more frequently it arises from the spread of inflammation from the nasal cavity (particularly in glanders) or from the alveoli of the molar teeth. But chronic catarrh of the mucous membrane concerned may be produced by a new growth, such as sarcoma, polypus, myxoma, botryomycoma, epithelioma or carcinoma, in the nasal cavity. Glanders also spreads sometimes to the mucous membrane of the superior maxillary sinus (antrum). Rickets may in rarer cases be the origin of the catarrh in foals.

**Pathological Aspect.**—At the outbreak of the complaint the mucous membranes of these accessory cavities are swollen, of a bluish or dark red colour and very highly charged with blood. Between the membranes and the bone we find a yellowish, gelatinous exudation; at the same time both bones and external skin are over-filled with blood. If the process be prolonged, the mucous membrane thickens and gives off a fluid, at first serous and later mucous and purulent, which, owing to the

swelling up of the orifice of discharge in the middle meatus of the nose, accumulates, thickens and frequently, also, decomposes (hydrops or empyema of the frontal and superior maxillary sinuses). Matter also collects sometimes between the mucous membrane and the bone, as a result of which the bone is wasted and thinned. In this way the external bony cover is bulged outwards and the cavity itself enlarged ; and the septum between the two divisions of the maxillary sinus may also become attenuated and even disappear entirely. In exceptional cases the catarrhal products of inflammation may even break through and form a fistula, or the pus may make itself a way to the dental alveoli and produce caries of the teeth. On the contrary a communication may be established between the cavities of the teeth and the sinus ; and one then sometimes finds the sinus stuffed with fodder. The enlargement of the frontal sinus may, moreover, lead to constriction of the brain-cavity with partial atrophy of the brain. In a similar manner the ethmoid cells and sphenoidal sinus are sometimes enlarged after a suppurative inflammation of their mucous membranes.

**Symptoms.**—The following are the morbid indications of catarrh of the sinuses of the head :

1. *Discharge from one nostril* : This is always the first sign observed. At the outset, if the inflammation be intense, this is sometimes mixed with traces of blood. Its consistence is various, as is also its amount. It usually presents a muco-purulent and thickish aspect, and is without special smell ; but may also be discoloured and malodorous. When the head is held down, it not unfrequently flows in larger quantity.

2. A *one-sided glandular swelling* in the intermaxillary space which, by new growth and induration of connective tissue, soon becomes hard.

3. *Bulging of the frontal or superior maxillary sinuses*. This usually somewhat diffuse expansion can be noticed by comparison with the sound side.

4. *Dulness of percussion-sounds* on the diseased side, owing to the collection of catarrhal secretions in the cavity and to thickening of the mucous membrane. We must observe, however, that when such secretions are only moderate, this dulness may be lacking, so that a negative result of percussion does not disprove the presence of catarrh in the superior maxillary sinus.

The *course* of the complaint is almost always chronic, and

may extend over months and years. As rare occurrences, brain symptoms may also appear, if the ichorous inflammation spread to the brain and its membranes, or if there be a suppurative inflammation of the ethmoid cells and sphenoidal sinus. Horses then present the aspect of sleepy staggers or brain inflammation.

**Diagnosis.**—Although it is not difficult to recognise catarrh of the sinuses from the indications given, there is yet another method of securing diagnosis, viz., trephining. This operation has a certain importance when it is desired to know if the catarrh be a simple one and not specific, or whether caused by glanders. When the nasal discharge and glandular swelling are on one side only, we are not seldom tempted to suspect glanders. The credit of introducing trephining as a method of differential diagnosis in such cases belongs to Haubner. Whereas, in simple catarrh the mucous membrane is, on trephining, often found to be smooth, in glanders it is frequently lumpy and uneven, and the glandorous process spreads readily to the flaps of skin formed by the operation. But even trephining does not give a positive result in all cases, for in simple, non-glandorous catarrh we not unfrequently perceive small, lumpy protuberances upon the mucous membrane. *Any doubts on this point may, however, quickly be removed by the use of mallein.* (See the differential diagnosis of Glanders, Vol. I., pp. 282-286.)

Sand has observed a mucous degeneration of the nasal conchæ in young horses, which in its morbid aspect closely resembles inflammation of the sinuses. The disease produces great expansion of the hollows of the nasal conchæ, of the upper jaw and of the frontal bone, causing muco-serous accumulations therein, which produce periodical nasal discharges, great distension of the neighbouring bones and difficulty of breathing.

**Therapeutics.**—A treatment of chronic catarrh of the sinuses is only successful when preceded by trephining; inhalations, as in chronic nasal catarrh, are useless. Trephining renders possible the unhindered outflow of the accumulated secretion, which, by decomposition, continually renews the inflammation, and also a direct treatment of the affected mucous membrane. It consequently always leads to cure, provided there be no more deeply seated changes, nor any new

growths. One may, by trephining, open up the higher division only, but it is better to operate between the two, so as to render both accessible; but occasionally it is advisable to trephine in two places, corresponding to the upper and lower divisions of the cavity. Through the opening made, inject solutions of creolin, lysol, sublimate, carbolic acid, sulphate of zinc, nitrate of silver, etc., and the openings must be kept unclosed until a complete cure is effected. It may not be out of place to suggest that the injections should be made if possible when the animal is standing, as when lying down some of the secretion may be drawn into the lungs and set up a broncho-pneumonia.

#### CHRONIC CATARRH OF THE GUTTURAL-POUCHES IN HORSES.

**Etiology.**—This is a somewhat rare condition. It usually occurs on one side, and only exceptionally on both. A primary affection of the mucous membranes of the guttural pouches scarcely ever happens, but catarrh usually spreads to them from some inflammatory process in the pharyngeal cavity, thus, in pharyngitis and strangles, or in consequence of the penetration of foreign bodies through the Eustachian tubes. How far congenital enlargement of the pouch is etiologically concerned therewith cannot be exactly settled. Chronic catarrh of the guttural pouch also occurs as an accompaniment of chronic glanders. Possibly an inflammation of the parotid glands may spread to the pouches. The usual chronic course of the disease arises from the difficulty of removing the exudation gathered in the pouch by way of the Eustachian tubes, and from consequent decomposition.

**Pathological Aspect.**—The mucous membrane, which at first is red and swollen, becomes thickened on long continuance, and consequently shows fibrous, nodular infiltrations and a lumpy surface, on which mould-fungi not unfrequently settle. The secreted matter is either tough and mucous, but more rarely of serous consistence or like pus, when the pus may thicken into caseous lumps. Sometimes, in consequence of the putrescent decomposition, it is ichorous, and the pouch distended by the gases thus generated (tympanites or meteorism of the guttural pouch). More rarely the pouch is found stuffed with fodder, which has entered either through the Eustachian tubes or through an opening made by an ulcer.

Finally, and when the process is very much prolonged, we have so-called chondroids in the pouch, i.e., thickened and variously degenerated masses of mucus and pus, of a hard and cartilaginous consistence, with round, often faceted surface, and ranging in size from an acorn to a chestnut or a goose-egg. In one case John found over 300 such concretions and Savarese about 250 in the guttural pouch of a horse.

**Symptoms.**—Chronic catarrh of the guttural pouch in horses manifests itself as follows:

1. By a one-sided (more rarely bi-lateral) nasal discharge of mucous, muco-purulent, purulent and lumpy character, which is sometimes also bad in colour and smell, and the head is sunk (in grazing, coughing) is frequently emitted in sudden rushes.
2. By swelling of the glands in the cavity between the branches of the lower jaw, such swelling being mostly on one side only.
3. By a swelling in the region of the parotid gland, which varies from fluctuating to doughy when pressed, or perhaps, owing to accumulation of air, is elastic and puffy. Sometimes on pressure it grows smaller, when the discharge is at once increased; and if charged with air (meteorism of the pouch) a tympanitic tone is emitted on percussion.
4. By difficulty of swallowing, a rattling respiration and even risk of suffocation when the pouch is much distended. In one case Dobesch observed haemorrhage from the catarrhally affected pouch which led to asphyxia. The course of the disease is chronic, and may last for years, and even for the animal's entire life.

**Diagnosis.**—This is in chronic cases not at all easy, and can only be made with certainty when a visible swelling of the pouch can be detected externally. Its similarity to the aspect of glanders is of the utmost importance, because the one-sided chronic discharge and one-sided glandular swelling both occur in the latter, so that for this reason the greatest caution is necessary. Still more easily can chronic catarrh of the pouch be mistaken for a simple chronic nasal catarrh, because the latter frequently accompanies the former; the same also with chronic catarrh of the sinuses, because in these sudden gushes of discharge through the nose are observed. Finally, swelling and abscess-formation in the sub-parotid

lymph-glands (upper cervical glands) may lead to the belief that we have a chronic catarrh of the pouch before us. The proposal to ensure diagnosis by introducing Günther's catheter (through the nasal cavity and Eustachian tubes) is not always easy to follow, nor always without danger. Thus Haubner saw the operation sever'l times result in pneumonia (due to foreign bodies), owing to the secretion having been inhaled into the lungs.

**Therapeutics.**—The treatment of chronic catarrh of the guttural pouch by internal remedies or inhalation is useless. It can only be cured by operation, i.e., by surgical opening and local application of astringent and disinfecting fluids. Respecting the method of such operation (hyovertebrotomy) consult the text-books on operative science and surgery.

**Tympanites of the Guttural Pouch.**—In addition to the tympanites which arises in the course of catarrh of the pouch from the development of gases of decomposition, there is also an abnormal accumulation of air resulting from the penetration of atmospheric air through the Eustachian tubes during the act of swallowing. We find this form of tympanites especially in foals, and it is attributed to anomalies of construction in these tubes which render possible the passage of air. Sometimes these air-accumulations cause much difficulty in breathing.

#### OESTRUS LARVÆ IN SHEEP.

#### GAD-FLY VERTIGO. SWINGING SICKNESS.

**Definition.**—The disease caused in sheep by oestrus larvæ arises from the presence of the larvæ of the sheep gad-fly, oestrus ovis, in the cavities of the nose and sinuses of the head, as well as in the hollow of the horns. This is revealed partly by symptoms of a chronic catarrh of one or all of the cavities named and partly by certain brain disturbances.

**Natural History Notes.**—The gad-fly of sheep is a small, yellowish-grey and almost naked fly, about  $\frac{1}{2}$  inch. in length and very common in Germany, which, particularly in hot and dry summers, settles in the crevices of sheep-folds, near where sheep pasture, on the edge of woods and in low bushes, and swarms in the hot noon-hours from July to September. The impregnated females then seek a flock of sheep, but on the approach of the flies the sheep become restless, run away or

hold their heads near to the ground, thrust their noses between their fore feet, or stand close together in a circle with heads depressed. Immediately after the larvæ have been deposited near their nostrils, the sheep shake their heads, run to and fro and rub their noses against hard objects or on their fore feet. But these signs of irritation soon pass away and for nine months the animals seem to be perfectly healthy.

The larvæ, which are probably already in that stage when deposited, creep into the cavities of nose and forehead, also into sinuses and the core of the horn, where after about nine months they reach maturity. Whereas at first they are very small and as fine as hairs, they attain a length when ripe of from  $\frac{1}{2}$  to  $1\frac{1}{2}$  inch, have in front two large, claw-shaped mouth-hooks, are yellowish-brown in colour, with darker transverse bands on the eleven rings which form the body. The mature larvæ emigrate in spring, from March to May (seldom earlier or later), and it is during this emigration that they produce the irritation of the mucous membranes of the nasal and accessory cavities and also of the brain known as œstrus sickness. In twenty-four hours from their emigration they change to the chrysalis state, and six to seven weeks later the perfect insect flies away.

**Post-mortem Lesions.**—In the cavities named we find a varying number (10 to 100) of the œstrus larvæ in different stages of development and surrounded by mucus and blood. The mucous membrane is very red, haemorrhagically infiltrated, much swollen, covered with mucus, pus and blood, and is even gangrenous. At the spot where the larvæ have bored their way in we find a round depression surmounted as by a wall. The cerebral membranes are hyperæmic and the brain is slightly oedematous. In rarer cases the larvæ have been known to enter the brain by piercing through the horizontal cribriform plate of the ethmoid. Some may even stray into the pharyngeal cavity, the larynx and trachea.

**Symptoms.**—These usually appear in spring as the œstrus larvæ begin to ripen, and consist first of all in a serous nasal discharge, which is later mucous and sometimes bloody; also in sneezing and snorting, whereby the larvæ which may then be emigrating are often cast out. At the same time the animals make shaking and swinging movements of the head (hence the name "swinging sickness"), rub their noses on the

fore leg or against some fixed object, so that they often become quite sore and excoriated, and the entire front of the head swollen to disfigurement. To this are added catarrhal inflammation of the conjunctiva, swelling of the eyelids and flow of tears. In slighter cases the disease is restricted to these symptoms. But in severer ones signs of depression are also developed on the part of the brain. The animals appear heavy in the head, have fits of dizziness (hence the name "gad-fly vertigo"), stagger, have a waddling gait, and occasionally twist to one side. This latter and much-disputed symptom does actually occur now and then (Gilos). If the disease take a fatal turn at this stage, as is usually the case, epileptiform spasms set in later, with grinding of the teeth, etc., and death then usually follows quickly, say within four to eight days, being sometimes marked by difficulty of breathing.

**Differential Diagnosis.**—Estrus-larval sickness may be especially confused with staggers, for which reason it was once known as "false staggers." But the catarrhal phenomena in the nose, the sneezing, discharge, shaking and swinging of the head, as well as the catarrhal conjunctivitis, all sufficiently assure diagnosis.

**Therapeutics**—Prophylaxis would best consist in keeping the sheep in their stable during the swarming season of the gad-flies. But as this is only rarely possible, one may at least try to protect them by rubbing the parts round the nose with tar, stinking animal-oil, creolin, etc., immediately before driving them out. But whether this does any good is doubtful. Any larvæ or chrysalis that may be found must be carefully destroyed. To prevent further immigration of the larvæ sneezing remedies have long been used (snuff, powdered hellebore, violet or carline roots), puffed into the nostrils with a quill. But this is only useful immediately after the larvæ have been deposited, and is later on of no use at all. When once the larvæ have penetrated to the cavities of the head they can only be removed by trephining. Zürn advises only to try this operation experimentally and on valuable animals, as it but rarely and exceptionally succeeds in removing all the larvæ. Injections are quite useless owing to the extraordinary power of resistance of the larvæ. The spot for the operation of trephining is in the upper angles of a cross formed by a line joining the two eyebrows and the median line of the head. The horn tips may

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also be sawn off. Early slaughtering is in all severe cases the best.

**PENTASTOMA TÆNIOIDES IN THE NOSE AND ACCESSORY CAVITIES OF DOGS.**

**Natural History Notes.**—The pentastoma tænioides (the tape-worm with five holes) is a worm-like creature usually classed with the acarinae. It was first discovered by Chabert in 1757, but its life-history was first revealed by the feeding experiments of Leuckart in 1856. He proved that the pentastoma denticulatum previously regarded as a separate species is only the larval form of pentastoma tænioides. Whereas the latter lives as a parasite in the breathing passages of flesh-eaters, particularly of dogs, the former has another host and inhabits the inner organs of herbivora.

1. *Pentastoma tænioides* is found most frequently in dogs and wolves, mostly in their nasal cavity and frontal sinus—as they have no superior maxillary—and especially in the sac-like enlargement of the central nasal passage, but also in the ethmoid cells, the pharyngeal cavity, in the larynx beneath the rima glottidis and in the middle ear. It is only rarely met with in other animals or in man. The parasite is from  $\frac{1}{2}$  in. to 5 in. in length (the female from 3 to 5 in. and the male from  $\frac{1}{2}$  to  $1\frac{1}{2}$  in.), is lancet-shaped, white or yellowish-white in colour, broad in front, pointed behind, and is frequently rolled up, having about ninety segments or rings, so that it resembles a tape-worm in appearance. The rounded head has a round mouth-opening on its lower side encircled by a chitinous ring, and near it four slit-like openings for its four legs, each of two members and furnished with a terminal claw, which legs can be drawn in at will. These slits were formerly taken for mouths, hence the name pentastoma or pentastomum, "having five holes."

2. *Pentastoma denticulatum* (*linguatula denticula*), the larval form of the above, is found most frequently in sheep, but also in oxen, hares, rabbits, deer, dromedaries, etc., and likewise in man. It occurs encysted in the liver, mesenteric glands, kidneys and lungs, and also free in the abdominal cavity and in the breathing passages. The larvæ are flat and elongated, white and transparent, about  $\frac{1}{16}$  inch long, and divided into some eighty segments, which are furnished with numerous spines (hence the name "denticulatum"). Their four or six feet are armed with double claws, and the sexual organs are rudimentary.

The process of development of *pentastoma tænioides* is as follows: The females living in the nasal cavities of carnivora lay about half a million eggs each, which issuing thence along with the nasal mucus upon the pastures, are there taken up by the grazing animals. The exterior husk of these eggs is dissolved in their stomachs, setting free a mite-like embryo,  $\frac{1}{5}$  inch long, with from four to six legs and provided with a tail. This embryo pierces the wall of the stomach, and makes its way to the parenchymatous organs, such as the mesenteric glands, liver, kidneys, etc., and there becomes encysted. In cattle the parasite occurs mostly in the mesenteric glands, less frequently in those of the pelvic and lumbar regions, but also in the liver and spleen (Ostertag). According to Babes, this pentastomum is in Roumania found incidentally in almost all oxen, and invariably in the mesenteric glands. During half a year the embryo undergoes several transformations and changes of skin, until it has attained the true larval form, when it quits the cyst and passes out into the abdominal cavity. Thence it works its way outwards through the lungs and bronchi, thus sometimes causing death from traumatic oedema of the lungs (Gerlach), or else is taken into the bodies of carnivora along with the flesh of the slaughtered hosts. According to Gerlach the *pentastoma* larvæ which pass out through the air-channels are very resistant, and may even dry up to a certain point without perishing. They reach the nasal cavities of carnivora either directly through the nostrils or from the pharyngeal cavity. After existing there for four to five months they become sexually ripe, and the *pentastoma denticulatum* has changed into *pentastoma tænioides*.

**Pathological Aspect.**—The changes wrought in the mucous membranes of the nasal and frontal cavities consist in swelling, redness, inflammation and even gangrene. The secreted matter is muco-purulent and not seldom bloody. It is remarkable that the *pentastoma* seems to be very common in some districts and very rare in others. Thus in Berlin we have very often found it, and only exceptionally in Munich. Hering only once met with the parasite in Stuttgart, while Colin found it in 64 Parisian dogs out of 630 examined, and from one to eleven specimens in each. In Berlin the parasite was only present in the larger breeds of dog, mastiffs and butchers' dogs. It was most frequently met with singly in the ethmoid cells, where it was firmly fixed by its mouth to the mucous membrane and

lamellæ of the bone. Owing to the depth to which it penetrates these cells and its similarity of colour to the nasal mucus, the creature may be easily overlooked, but it can be best recognised by the narrow, pale grey longitudinal stripe of its intestinal canal. In most cases the pathological changes wrought in the nasal cavity were not serious. They consisted in swelling, epithelial desquamation, redness and mucous disintegration of the mucous membrane. In one case there was also haemorrhagic inflammation of the ethmoid cells. In a dog which had suffered a month from chronic nasal discharge the presence in the nasal mucus of pentastomum eggs could be demonstrated.

**Symptom.**—The parasites may exist in the nasal cavities almost without external sign. But very frequently they produce the symptoms of chronic nasal catarrh. The animals sneeze, discharge from the nose, wheeze in breathing, rub their noses on hard objects or against their feet, sometimes sneeze or snort out a few pentastomata, occasionally bleed from the nose and may have difficulty of breathing, and even fits of choking. At times the sense of smell seems to be entirely lost. The animals do not eat their food from the ground, but only when it is held before them, and are often noticed to grow rapidly thin. As a peculiar complication in a few cases, perforation of the hard palate has been observed, causing great flow of saliva (Adam, Perdan). Usually the trouble ends after a considerable time in ejection of the pentastoma and in consequent recovery.

In other cases the above signs are complicated with cerebral phenomena, which sometimes cannot be distinguished from hydrophobia. The animals are restless, excited, irritated, snapish, roam about the streets, bite readily, howl loudly and continuously, burrow into their straw, thrust their heads between the bars of their cage and manifest rotary movements, or else seem from the outset to be stupefied and quite apathetic. Occasionally paralysis of the lower jaw has been noticed. In such cases death generally supervenes in a few days. On *post-mortem* we find signs of hyperæmia, also of oedema and slight inflammation in the brain. Of diagnostic importance is the identification of pentastomum eggs in the nasal discharge.

**Pentastomum in Horses.**—In a case published by Schwammel five specimens of pentastoma tænioides were found in the nasal cavity of a horse slaughtered on suspicion of glanders. The animal had a mucopurulent nasal discharge and glandular swellings, frequently snorted and shook its head.

**Differential Diagnosis.**—More important than any confusion with distemper or a simple nasal catarrh is the distinction between pentastoma sickness and rabies. This is only possible when the parasite can be discovered during life or by a *post-mortem*. It is therefore indispensable, owing to the great similarity of many of their symptoms, to carefully examine the nasal and frontal cavities on dissection, to see if there be any pentastomata, *i.e.*, should there be reason to suspect rabies. We must add in this connection that some observations seem to show (Friedberger, Perdan) that these parasites may cause serious brain disorder and even death without producing any marked changes on the nasal and frontal mucous membranes.

**Therapeutics.**—A radical cure can only be expected from trephining the nasal and frontal cavities. An opening into the latter can of itself do no harm even if the parasite be lodged in the other cavity, as injections of suitable remedies (creolin, benzol, carbolic acid, etc.) can be made from the frontal sinus. Mere inhalation of tar-vapour, or the use of sneezing remedies is of little or no use.

**Pentastoma Denticulatum** is said to produce symptoms of peritonitis, inflammation of the bowels, etc., in rabbits (Zürn). Except in the experiments in feeding carried out by Leuckart, no injurious effects have been observed in the herbivora infested by these parasites. Leuckart in his tests found serious destructive changes in the lungs and liver of the hares experimented on, consisting in haemorrhage, inflammation and greatly ramified, haemorrhagic passages through the organs named, etc. For information on the changes effected by the parasite in the mesenteric glands of cattle, and also concerning the importance of pentastoma from an official meat-inspector's point of view, see Ostertag's work on Meat Inspection.

## CHAPTER XXII.

### DISEASES OF THE LARYNX.

LARYNGITIS. INFLAMMATION OF THE MUCOUS MEMBRANES OF THE LARYNX.

**Classification.**—Inflammatory conditions of the laryngeal mucous membranes often exist alone. But not seldom the neighbouring organs also participate, especially the mucous membranes of the pharynx, nasal cavity and trachea. In such less definitely localised cases the term "laryngitis" can only be used when the symptoms are most pronounced in that organ. When this is not the case, we should speak rather of "catarrh or inflammation" of the upper air-passages. As in other inflammations of mucous membranes, we have to distinguish various degrees of intensity. Thus we divide laryngitis into catarrhal, phlegmonous, croupous, diphtheritic and ulcerative. In accordance with its causes, we may also speak of it as traumatic, rheumatic, infective, tuberculous, glandular or sympathetic; and from its course, as acute and chronic. We content ourselves with discussing the three following classifications :

1. Acute catarrh of the larynx.
2. Chronic catarrh of the larynx.
3. Croupous and diphtheritic inflammation of the larynx.

I. ACUTE CATARRH OF THE LARYNX. LARYNGITIS CATARRHALIS ACUTA.

**Occurrence.**—The acute catarrh of the larynx known ordinarily as a swollen throat or sore throat, is very common in horses and dogs, they being more exposed to injurious external influences than other domestic animals. Among the dogs treated in the Berlin dog-hospital three per cent. are afflicted with laryngitis.

**Etiology.**—The cause of primary independent catarrh of the larynx is in most cases cold. The inhalation of cold air, drinking very cold water, or severe chill of the skin by heavy rain, cold winds and draughts, etc., very soon cause catarrhal laryngitis, especially in pampered or delicate animals. Hence the almost epidemic appearance of the complaint in spring or autumn. Traumatic and chemical agencies acting on the mucous membranes of the larynx are further sources of the trouble, also external pressure on the larynx, persistent barking in dogs, irritation caused by foreign objects, drugs, smoke, pungent vapours, especially those of chlorine and sulphur. Also the so-called "mash-cough," which occurs in cattle on being fed for the first time on potato mashes, seems to have a similar origin, as probably certain substances contained therein, especially alcohol, exert an irritating influence upon the laryngeal mucous membrane, to which by degrees the animal accustoms itself.

Acute laryngitis occurs secondarily as an accompaniment of various general diseases, such as glanders, distemper, pleuropneumonia, malignant catarrh, etc. Moreover, a catarrhal process often spreads from the cavities of the nose and pharynx, the trachea and bronchi to the larynx. Information on infectious laryngeal catarrh, appearing epizootically among horses and cattle, will be found further on in this chapter.

**Pathological Aspect.**—The pathological changes consist in swelling, redness and even ecchymosis of the mucous membrane, and in accumulation of a serous, mucous or purulent exudation. According to Brückmüller, it is mainly the upper part of the larynx which is affected, and the laryngitis may sometimes develop to a suppurative destruction of the mucous glands and superficial ulceration of the epiglottis and vocal cords.

**Symptoms.**—The chief sign of acute laryngeal catarrh is cough. This is at first dry and rough as well as painful, and is either short and broken or else prolonged. Later, it becomes looser and accompanied by a mucous sputum, which dogs at once swallow again. This cough is easily excited by bringing the animals into the open, by eating and drinking, by rising quickly or leaping, by strong excitement, e.g., when dogs in hospital are visited by their owners. The catarrh also manifests its presence by extreme sensitiveness of the laryngeal

region to pressure, which the animals try to evade and which often produces paroxysms of coughing. Owing to great swelling of the mucous membranes and oedematous infiltration of the crico-arytenoideus posticus, breathing is also impeded, and one may sometimes hear from a distance a rattling, whistling or wheezing sound, which on auscultation of the larynx may be distinctly heard along with other noises, such as rhonchi. In horses the head is frequently held somewhat stretched out. In addition to these most important signs of catarrh of the larynx, we notice when the condition is prolonged, the symptoms of pharyngitis, bronchitis, or rhinitis, diminished appetite, impeded swallowing, dryness of the mouth, nasal discharge and sometimes slight fever. The latter is always referable to complication with one of the other symptoms named. A higher degree of fever always awakes suspicion of a graver, especially an infectious disease.

**Differential Diagnosis.**—The chief symptom of catarrhal laryngitis, viz., the cough, occurs not only in the other inflammations of the larynx, but also in a further series of diseases of the breathing apparatus, such as tracheitis, bronchitis, pneumonia and pleuritis. Diagnosis cannot therefore be based on the cough alone, we must note also the other signs, such as increased sensitiveness of the larynx to pressure and the abnormal laryngeal breathing sounds. Tracheitis and bronchitis are essentially characterised by the presence of tracheal or bronchial rattling sounds; pneumonia and pleuritis are diagnosed by physical examination of the lungs or thoracic wall. A differentiation between simple and severe inflammatory processes in the larynx is not always very easy. In general we attempt definite diagnosis from the severity of local symptoms, the degree of fever, the gravity of the general malady and, above all, from the nature of any matter which may be coughed up, as well as from certain characteristic breathing sounds.

**Therapeutics.**—In acute laryngeal catarrh prophylaxis consists in hardening the animals and accustoming them to all degrees of weather. Against the disease itself we recommend rest and avoidance of hard labour, especially in unfavourable weather; moreover, good ventilation must be provided. The application of moist warmth to the region of the larynx is the general practice, viz., in the form of Priessnitz's compresses, and the results are often very good. Inhalation

of steam is also often tried. The cough may also be combatted by internal administration of narcotics, especially morphia and codeine. Sometimes the latter seems to be more effective than morphia.

**Epizootic Laryngo-tracheal Catarrh in Horses.**—In the summer of 1888 an epizootic catarrh of the upper air-passages raged widely throughout all Germany, in which twenty regiments in ten different army-corps in Prussia alone were attacked, but which also occurred in private stables. This broke out again in 1893, but not so universally. The disease was remarkably contagious, and not rarely all the horses of a stable or of a division fell ill in a few days. Infection was transmitted by the exhaled air, for horses were smitten through being merely led past a sick animal at a few paces' distance. It also attacked impartially horses which had already survived murrain, pleuro-pneumonia or influenza. The incubation stage only lasted a few days. Its chief symptom was in all cases a dry, strong, sharp cough (seldom faint or painful), as well as great sensitiveness of the larynx and trachea to palpation, which operation at once brought on a violent coughing fit. As the disease advanced, a very slight nasal discharge usually began, at first watery and afterwards mucous, complicated sometimes with a slight swelling of the submaxillary lymphatic glands. Auscultation and percussion of the lungs gave, as a rule, no abnormal result. In some of the outbreaks (1893) conjunctivitis also set in. Fever was not regularly present, but occurred sometimes during the first few days of the malady, commencing then sometimes with shivering fits lasting for hours, but only occasionally did the temperature reach 106° Fahr. Rust says that among 46 sick horses the temperature of 103 exceeded 102°. Only in the most feverish cases was appetite disturbed, the breathing quickened to twenty to twenty-four, the sensorium dulled or bodily force abated. In the immense majority of cases the course was very benign, the illness not lasting more than eight to fourteen days, and after about a week of convalescence the horses could return to work. More rarely this period lasted longer. Only occasionally, and when the sick animals were put to severe labour or exposed to sharp cold, were complications observed with colic, intestinal catarrh, pneumonia or pleuritis. The *treatment* was purely dietetic: avoidance of severe bodily exertion, moderate exercise in the open. Some observers (Zürn) recommended artificial infection of all the threatened horses as a means of shortening the epidemic. This latter is now regarded as a peculiar and hitherto little known infectious disease, which was formerly very likely included under the name of "influenza in its wider sense." But many of the descriptions have an undeniable resemblance to murrain (the contagious pleuro-pneumonia of horses) and influenza (the distemper of horses), and a careful examination would be well undertaken to enable us more clearly to discriminate between these various diseases.

**Epizootic Catarrh of the Air-passages in Cattle.**—Also in cattle the appearance of an enzootic and even epizootic outbreak of catarrhal affections of the air-passages has at times been observed. Such an outbreak occurred in Bavaria in 1890. The disease was ac-

accompanied by high fever and ran a pretty regular course (rhinitis, laryngitis, tracheitis and bronchitis). It was by many connected with the influenza (*la grippe*) then raging among men. Its chief symptoms were: mucous nasal discharge, frequent and painful fits of coughing, redness of the nasal mucous membrane, great sensitiveness of the larynx to pressure, dyspnoea, increased vesicular respiration and rattling sounds in the lungs, diminished appetite, high fever (104-107° Fahr.), shiverings, tripping, stumbling, etc. In a few cases there was also conjunctivitis catarrhalis. The severest cases usually lasted only a few days, their total duration being eight days, and occasionally two to three weeks. The phenomena were in almost all animals alike, and so also was the thoroughly typical course. Several animals in one byre always sickened together. In solitary cases the appearance of catarrhal pneumonia was noted as a complication, which was manifested by irregular dulness to percussion and bronchial breathing. In such single instances the complaint led to confusion with pleuro-pneumonia, and also, owing to the catarrhal affection of the tarsal conjunctiva, with malignant catarrhal fever. The course was in general very benign. Owing to the typical process of the disease, treatment was seldom required. As regards the connection between this disease and the influenza of man, fuller proof is lacking, although the possibility of such connection cannot be denied.

## 2. CHRONIC CATARRH OF THE LARYNX. LARYNGITIS CATARRHALIS CHRONICA.

**Etiology.**—Chronic catarrh of the larynx, or, as it is commonly called, spasmodic cough, or irritation cough, occurs oftenest in horses and dogs, and is very widespread in spring and autumn. Among dogs, older and long-haired terriers suffer most, also Pomeranians, pugs, poodles and spaniels. Of horses, those used for riding and better-class carriage-work suffer most frequently. The causes are essentially those given for acute catarrh, out of which, in fact, most chronic cases develop. Cold plays the chief part, which accounts for the almost epidemic character of the disease in spring and autumn. Chronic conditions of irritation may also arise in the larynx from traumatic causes, or may remain behind after acute infectious disease; thus in horses after pleuro-pneumonia and strangles, or in dogs after distemper. A further source of chronic laryngitis is found in tumours arising in the larynx, such as papillomatous proliferations, polypi, sarcomata, carcinomata, actinomycomata, glanderous and tuberculous new growths which maintain a chronic catarrh of their surroundings. In one case Nocard observed chronic cough, or catarrh of the larynx, in a dog, resulting from pressure of a tumour of the mediastinum upon the vagus.

**Whooping Cough in Man.**—We cannot share the opinion expressed by Röll and others that the so-called spasmodic or inflammation cough

of horses and dogs is identical with the whooping cough (*tussis convulsiva*) of man, because there are no proofs for this assumption. Whooping cough in man represents a contagious, infective disease, which, according to some, is to be regarded as mycosis of the mucous membranes of the larynx and wind-pipe, and, according to others, as neurosis of the larynx superior, which especially attacks children, which never recurs when once it has been passed through, and is marked by paroxysmal fits of coughing with intervals of quiescence. All these characteristic signs are absent in the spasmodic cough of animals. It is not contagious, attacks chiefly older animals, is remarkable for its frequent recurrence and has no typical course.

**Pathological Aspect.**—The pathological changes in chronic catarrh of the larynx are precisely the same as on other mucous membranes. In consequence of chronic hyperæmia the blood-vessels are permanently dilated, and the mucous membrane is thickened and uneven. According to the locality, papillary protuberances (proliferation of the papillary body), or very small nodular irregularities lie upon its surface, giving it a granulated appearance (enlargement of the mucous glands, laryngitis granulosa). The epithelium, being cast off more freely, forms white patches upon the membrane; the connective tissue is hypertrophic and permeated by white blood-corpuscles. But in dogs we have often, very strangely, failed on superficial examination to find any visible changes of the mucous membrane.

**Symptoms.**—The most important and often the only symptom is a cough. This is usually dry, rough and croaking, but more rarely is moist and combined with a humming or rattling sound. Frequently there is at the same time a certain degree of dyspnoea. In horses the cough often occurs in fits, with hard stretching of the head and neck. In immediate connection with the cough in dogs we often notice spasmodic choking, the neck being stretched and the head held close to the ground, and this is usually combined with vomiting. This choking and vomiting are caused by the tough, thick mucus coughed up from the larynx into the pharynx. It is also characteristic that in dogs the coughing fits occur with greater severity by night, which evidently arises from the gradual accumulation of this mucus. General condition is not disturbed. The animals are otherwise quite lively, free from fever, their appetite is normal, breathing unimpeded, and auscultation or percussion of the lungs yields negative results. The duration of chronic catarrh of the larynx is mostly somewhat long, and extends

over weeks, months, and even years. In this respect, and owing to numerous relapses, the prognosis of the disease is very unfavourable.

**Therapeutics.**—Here great quiet and the avoidance of all excitement are of great importance, as every attack of coughing only increases the hyperæmia of the laryngeal membranes. We also recommend hydropathic wrappings in the form of Priesanitz's compresses for the throat. Derivatory inunctions of the skin round about the larynx (tartar-emetic ointment) were formerly much used. But the matter of most therapeutic importance is local treatment of the affected membranes. Although not such quite good results are obtainable here as from similar treatment in man, yet this method is by far the best. It consists in the inhalation of vapour (steam, aromatic flowers, creolin, carbolic acid, tar or oil of turpentine; 1 per cent. solutions of sodium chloride, ammonium chloride, bromide of potassium, tannin or nitrate of silver); in insufflating powder into the larynx (morphia, nitrate of silver,  $\frac{1}{2}$  grains); sugar (15 grains); also in painting the mucous membrane with a brush or small sponge. The liquid specially used for this purpose is a 1 to 5 per cent. solution of nitrate of silver. For inhalation a sac-like mask is used for horses, but in case of necessity this may be replaced by a rug held over the head. The inhaling apparatus used for man may be utilised for dogs also, or large inhalation chests, such as are customary in veterinary hospitals, may be used. For horses Dieckerhoff recommends the direct injection of astringent fluids into the larynx. He uses a bent, hollow needle, which, in horses, he pushes into the crico-tracheal ligament and twists upwards towards the larynx. The fluids used are: a  $\frac{1}{2}$  per cent. solution of alum, a  $\frac{1}{2}$  per cent. solution of sugar of lead and a 5 per cent. mixture of subnitrate bismuth. We can to some extent confirm the efficacy of these injections, although their application is not quite without danger. In dogs we make the injection through the crico-thyroid ligament.

The internal administration of remedies to allay the cough is much less efficacious than direct treatment, but may appropriately be combined therewith, especially for dogs. For them we generally prescribe morphia in conjunction with prussic acid or codein. To horses one may give expectorant powders upon their fodder, e.g., Rx sal-ammoniac  $1\frac{1}{2}$  oz.; common salt; pulv. liquorice,  $\frac{1}{2}$  3 oz. Sprinkle a tablespoonful on each feed.

An addition of bromide of potassium or iodide of potassium increases the efficacy of the latter recipe.

### 3. CROUPOUS AND DIPHTHERITIC INFLAMMATION OF THE LARYNX.

**General Notes.**—Croupy laryngitis, which is also known as laryngeal-angina, croup or angina, does not in most cases confine itself to the larynx, but the croupous process usually attacks at the same time the trachea as far as the bifurcation of the bronchi. The inflammation also spreads very often to the pharynx, as happens reversely in pharyngitis. Nevertheless, in laryngeal-angina the larynx is the primary and most important seat of disease, whereas in pharyngeal-angina this applies to the mucous membranes of the pharynx.

**Occurrence.**—Laryngeal-croup is commonest in cattle and next to them in horses and cats. Other kinds of animals suffer much more rarely.

**Etiology.**—This croupy inflammation of the laryngeal membranes seems to arise most frequently from cold. In this respect very rapid changes of temperature, especially in mountainous regions, passing the night out of doors and the consumption of very cold food and drink are chiefly blamed. Formeris states that the ailment is particularly rife in the mountains of Piedmont. Along with cold we must also cite traumatic injuries of the membranes of the larynx as a cause of croup, also the action of foreign bodies, of heated air, smoke, pungent infusions, among which ammonia is particularly dangerous, also of irritant gases, such as vapour of chlorine, or of sulphuric acid, etc. Occasionally, owing to the enzootic appearance of the malady, it almost seems to be of infectious origin. Diphtheritic affections of the laryngeal mucous membranes, i.e., necrotic processes in the tissue of the membrane itself, with formation of ulcers, produce sometimes an extreme degree of croupous inflammation. In other cases we meet with it as a secondary phenomenon in the course of certain infectious diseases, such as the malignant catarrh of cattle, petechial fever and acute glanders.

**Pathology.**—The changes wrought by laryngeal croup upon the mucous membranes of the larynx and trachea consist at first in an insular deposit like hoar-frost, which later

becomes confluent and gradually thickens to a grey or whitish-yellow (in cattle even yellowish-brown) membranous coating, which often forms perfect casts of the larynx and trachea and is sometimes easy, sometimes difficult to dislodge. The microscope shows this coating to consist mainly of fibrinous threads or stalks, white blood-corpuscles as well as free nuclei. The subjacent membrane is hyperæmic and even haemorrhagic, being also swollen and infiltrated with pus. The sub-mucosa is frequently in a state of œdema. In diphtheritis of the larynx we often find discoloured membranous deposits, greyish-white to greyish-yellow with a tinge of green, which leave ulcerous losses of substance of the mucous membrane behind them, and sometimes spread forwards in the pharynx and to the root of the tongue, and downwards to the trachea and bronchi. As complications of croupous or diphtheritic laryngitis we must indicate : tracheitis, bronchitis, croupy pneumonia, the pneumonia due to foreign bodies, pulmonary emphysema.

**Symptoms.**—Laryngeal croup signalises itself by the rapid and sudden appearance of severe illness. General condition is from the first gravely compromised, the disease often setting in with very high fever and shivering fits. Then amid violent attacks of coughing much difficulty of breathing develops. The animals breathe more or less dyspnoeically with considerable distension of mouth and nostrils, stretch out their heads, slaver profusely, sometimes press forward against the bar, or stand anguish-stricken with outspread forelegs, their mucous membranes being very red, their eyes prominent and glaring and the veins of the skin charged to repletion. The region of the larynx is swollen, hot and sensitive to pressure ; indeed, a sharp pressure of either larynx or trachea may cause risk of suffocation. Meanwhile each respiration is accompanied by the most varied sounds : groaning, whistling, rattling, flapping, buzzing ; and on laying the hand over the larynx one can distinctly feel a laryngeal fremitus. If these symptoms rapidly increase, death may follow from asphyxia in a few days. If, on the other hand, the malady take a milder course, then after from three to five days membranous croup-shreds or tubular casts of the larynx and trachea begin to be coughed up through the mouth and nose, after which recovery quickly follows, and the entire duration of the sickness will not exceed five to six days. Among other accompanying symptoms are : suspension of eating and rumination, salivation and regurgitation, constipation and cessation of

the milk-yield. Sometimes the animals perish from subsequent inflammation of the lungs. The aspect of laryngeal diphtheritis is similar, though mostly more intense.

**Prognosis.**—In cattle and horses the prognosis must at best be termed doubtful, as suffocation may occur at any moment. But recovery may come as rapidly as death. A forecast seems to be more favourable in cats, for we have several times seen the complaint with them run a perfectly mild course.

**Therapeutics.**—The treatment of laryngeal-angina is, owing to its very rapid course, somewhat precarious. Usually we apply inhalations of steam or lime-water, Priessnitz' compresses, continuous hot poultices, friction with quicksilver ointment, and also astringent fluids, such as solution of nitrate of silver. Trasbôt recommends bleeding cattle, with subsequent administration of iodide of potassium in daily doses of  $2\frac{1}{2}$  drams. The most important point in treatment is that there be no delay in performing tracheotomy if the dyspnoea become severe; very often this is done too late. For cats and dogs an emetic may be tried.

#### ŒDEMA OF THE GLOTTIS. ŒDEMA OF THE LARYNGEAL MUCOUS MEMBRANE.

**Definition.**—Under the name of œdema of the glottis we describe an œdematosus swelling of the sub-mucous connective tissue in the larynx, which sometimes exhibits the characters of a serous or suppurative inflammation (phlegmon), and sometimes that of a true œdema due to obstruction.

**Etiology.**—1. An *inflammatory* œdema of the glottis either arises primarily in the course of intense laryngeal inflammation after injury of its mucous membranes by foreign objects or pungent drugs, as well as after cold, e.g., under rapid changes of temperature after a previous laborious march through much dust; or else it arises secondarily as a collateral œdema in connection with the inflammation of some adjoining membrane, such as a severe pharyngitis, also in the course of many infectious diseases, e.g., anthrax, glanders, petechial fever, pox, pyæmia or pneumonia.

2. *True congestive œdema* of the larynx arises from congestion of blood such as follows heart, lung or kidney diseases, or from

compression of the jugulars. We have repeatedly seen it in horses after previous strangulation.

**Pathological Aspect**—The œdema generally attacks only the upper portion of the larynx, i.e., the base of the epiglottis, the arytenoid cartilage, epiglottic ligaments and the ventricular ligament. The lower parts, and especially the rima glottidis with its ligaments, are seldom affected, because there the mucous membranes are more firmly attached to their base. The œdematosely swollen mucous membrane forms thick, gelatinous, quivering blisters at the spots named, and particularly on the vocal chords, which project into the orifice of the larynx. These blisters or swellings are sometimes pale and at others very red. On being cut they discharge, according to the nature of the swelling, a serous, purulent or gelatinous fluid. Occasionally the adjoining parts of the pharynx, root of the tongue and nasal cavity are found to be œdematosely.

**Symptoms.**—1. The phenomena of *inflammatory* œdema of the glottis appear very rapidly and suddenly. Its aspect, which is closely related to that of croup, consists essentially in extreme difficulty of breathing, leading finally to asphyxia, in which inspiration is more especially impeded. The animals breathe dyspnœically with much panting, groaning, rattling, and often with a whistling sound which can be heard from a distance. They cough, display fear, outbreaks of sweating, injected mucous membranes, protuding eyeballs, and, rapidly perish in a few hours if help be not speedily afforded.

2. In *true congestive* œdema the course is slower, and the severe stages only occur after several days or weeks.

**Diagnosis.**—The frequent confusion of œdema of the glottis with laryngeal croup is easily explained by the great similarity of their symptoms. But the diagnosis of the former is assured by its frequent peracute course, and by the fact that no fragments of membrane are coughed up.

**Therapeutics.**—The only sure remedy in œdema of the glottis, when blisters in the region of the larynx have proved unavailing, is the performance of tracheotomy with all possible speed. Other manipulations, such as blood-letting, the use of strong purgatives, the administration of ice-pills, the inhalation of or painting with astringents, may be

useful in sub-acute cases, but are not much good in those of rapid course.

**ROARING. WHISTLING. HEMIPLLEGIA LARYNGIS. STRIDOR LARYNGIS. ATROPHY OF THE CRICO-ARYTENOIDEUS POSTICUS, LEFT-SIDED PARALYSIS OF THE VOCAL CHORD.**

**Occurrence.**—Left-sided paralysis of the vocal chord due to paralysis of the recurrent laryngeal nerve, *i.e.*, left-sided atrophy of the crico-arytenoideus posticus, and, indeed, of the posterior crico-arytenoid muscle, as well as of the left half of the transverse arytenoid muscle, is a disease peculiar to horses in their younger years (three to six). It was first etiologically investigated in 1823 by Günther the elder, and described in 1834 in a classical treatise. The complaint, commonly known as "whistling" or "roaring," is of the greatest scientific interest both clinically and forensically, but we can here only treat it from the former standpoint. This recurrent paralysis has in a few cases been observed in cattle and dogs, as well as in horses. In Europe laryngeal whistling is commonest among the nobler breeds of England, France, Hanover and Holstein. But the English thoroughbred is of all horses most subject to it. On the other hand, the ailment is exceedingly rare in India, Egypt, the Cape, Australia, South America, and especially in Arabia (Fleming). Stallions suffer much more frequently than mares (Möller).

**Etiology.**—The causes of this defect are in most cases to be sought in heredity. More especially do the finer English horses, and particularly stallions, transmit it to their issue, a fact severely felt in some breeding studs. Horses with long, thin necks seem most inclined to it. But the ailment is by no means confined to English thoroughbreds, but occurs in all equine races and especially among English and German half-breeds. Many trace its origin to the methods of training English racehorses, which greatly develop the heart and aorta, so that, owing to the extreme leanness of all parts, the recurrent laryngeal nerve is much more exposed to the pulsations of the aorta. Steamy, badly-ventilated stables, and the disposition to catarrh thereby produced, are also blamed as indirect causes.

The question has often been asked why in the vast majority of cases the muscular atrophy is only on the left side. According to Günther, out of 100 cases, 96 are left-sided; Fleming

says 99 and Cadiot 95. Frequently the superficial course of the left recurrent laryngeal nerve in the neck has been blamed. But more important for the settlement of this question seems to be its course in the thoracic cavity, to which Günther first called attention. The left recurrent laryngeal nerve covers a greater distance in the thorax, because it winds round the aortal arch. Günther was the first to observe that in the course of frequent chest diseases of horses (pleuro-pneumonia) the left laryngeal nerve is also affected and produces a left-sided paralysis of the larynx. Franc held that the nerve is overstrained by the aorta, which is so highly developed in thoroughbreds, and to this opinion Martin also adhered. Martin points especially to the embryological side of the question. He believes that the recurrent laryngeal nerve is irritated by the arterial arches, the alternating motion of the heart and the great length of the neck in thoroughbred horses. The constricted position of the recurrent between the aorta and the trachea is also etiologically blamed (Vaerst). According to Sussdorf, the nerve in question is always visibly flattened at the spot where it passes between the aorta and the wind-pipe.

A relatively frequent cause of roaring is also murrain or pleuro-pneumonia, from which it remains behind as a sequel. Whether the development of roaring in such cases is connected with an inflammatory affection of the recurrent nerve, which has spread from the inflamed pleura to the nerves, or with some other disturbance of the nervous system (paralysis of the nerves by the action of poison), cannot at present be decided.

We sometimes also find that compression and paralysis of the recurrent laryngeal nerve in its course along the neck are effected by tumours, e.g., by lymphomata, struma, abscesses of the lymph-glands during strangles, dilatation of the pharynx, and also in the thoracic cavity by enlarged bronchial glands. It has been experimentally shown that accidental injury of the recurrens at this spot produces the same effect.

Finally, in the course of serious phlegmonous inflammation of the pharynx and larynx (strangles, angina) a muscular inflammation and atrophy of the larynx occurs, with stenosis of the rima glottidis, in which either a paralysis of the muscles is developed from the very first, or else, later on, a chronic sclerotic inflammation of the connective tissue of the sub-mucosa, with stenosis of the space between the two arytenoid cartilages. Poison may also result in laryngeal whistling, owing to nervous or muscular paralysis. In this connection we must mention

chronic lead-poisoning, the eating of chick-peas, mutters, tares and lucerne, which perhaps effect this curious result owing to the presence of certain fungi. According to Fleming, from 2 to 4 per cent. of all cases of this malady are traceable to these vegetable poisons.

**NOTE.**—According to recent researches of Thomassen, of Utrecht, 95 per cent. of cases of roaring are neuropathic in origin, and in 99 per cent. of cases of nervous roaring there is marked degeneration in the peripheral portion of the left recurrent laryngeal nerve, viz., in the region of the larynx. The thoracic portions of the nerve always remained normal even in those cases in which roaring had existed for several years, also when roaring followed strangles or pleuro-pneumonia the thoracic portion remained sound. In not a single instance could any change be detected in the nerve where it doubled round the aorta.—(J. D.)

**Pathological Aspect.**—The muscles on the left side of the larynx which expand the rima glottidis are atrophied and fatty-degenerated, and are noticeable for their pale, yellowish-red colour and their similarity to stripes of connective tissue (prominence of the perimysium). The atrophy is usually, however, not complete, but affects mainly the posterior crico-arytenoid muscles. This muscular atrophy corresponds almost always exactly with the area over which the recurrent nerve extends. This nerve itself is also often found atrophied and yellowish (fatty degeneration). In rare cases the atrophy is on the right side; but when there has been previous severe laryngitis, it is mostly total. In contrast to atrophy of the other laryngeal muscles, the crico-thyroid muscle is in roaring frequently found to be much developed by way of compensation. The direct consequence of paralysis of the muscles of the larynx is an obliquity of the left arytenoid muscle, which sinks downward, and thus narrows the entrance to the larynx. The lungs are sometimes found in a condition of emphysema.

**Symptoms.**—The main symptom in paralysis of the recurrent nerve consists in an *inspiratory* sound originating in the larynx, which arises from the arytenoid cartilage of the affected side sinking towards the interior of the laryngeal cavity, in consequence of which the rima glottidis can no longer be sufficiently opened, owing to paralysis of the left vocal chord. This breathing sound (stridor) can only seldom be heard when the animal is at rest, as in eating (these are extreme cases), but becomes audible on movement, and is characterised sometimes as whistling or piping, at others as rattling and snoring, snorting,

panting and wheezing, or, again, as croaking, screeching, bellowing or roaring. The sound is often so intense as to be heard a long way off. It is usually only emitted during inspiration, and only in severe cases during expiration as well. The nostrils are then always widely dilated like a trumpet. The noise is almost invariably loudest after severe exertion, and especially after galloping, but dies away of itself after a few minutes' rest. It is characteristic that the stridor may be momentarily silenced by pressing the dilated nostrils together, so that only a small stream of air can enter, corresponding to the narrowed rima glottidis. In slight cases the defect can only be observed after forced exertion. As the breathing sounds grow in loudness, so also does the animal's difficulty of breathing, which may at times amount to choking fits and collapse. The buzzing or quivering cough is also remarkable, as during the inspiration which follows each violent expiration the relaxed left vocal chord vibrates or quivers. In the same way in roarers the neigh is sometimes hoarse.

A further guide in the proof of muscular atrophy may be found in palpation of the larynx. In advanced stages of the complaint the left arytenoid cartilage can be pressed in more deeply than the right when the larynx is grasped by the fingers; moreover, even when at rest, the characteristic laryngeal sounds may be thus set up. According to Bassi, on examining the interior of the larynx, after introducing a gag, one may observe a displacement of one arytenoid cartilage as well as relaxation and obliquity of the vocal chord. But this attempt does not always succeed, and it is more important to try an endoscopic examination of the inside of the larynx, and especially of the vocal chords, by Polansky and Schindelka's laryngoscope, a very valuable instrument for this and other purposes.

The course of the condition is generally chronic, and, indeed, it usually tends to grow worse. Roaring develops frequently in the course of acute diseases, especially of pleuro-pneumonia. Cure can only be effected when there is paresis of the laryngeal muscles (partial paralysis), never when the paralysis is complete. Recovery is most probable when the muscular paralysis is a sequel of pleuro-pneumonia or severe laryngitis, and in the toxic form may be secured by avoidance of the offending fodder. The nature of the service required from the sick animals has also great influence upon the course of the condition.

**Differential Diagnosis.**—Left-sided paralysis of the vocal chords may be confused with roaring caused by other mechanical contractions of the air-passages, such, for instance, as occur in the course of stenosis of the nostrils, posterior nares, pharyngeal cavity and larynx, or of the trachea as a result of new growths, polypi, cysts, congenital deformities, impressions, fractures, swelling of the guttural-pouch, operations (tracheotomy), etc. In all such cases palpation of the larynx will decide the question, or, if still doubtful, an examination by the laryngoscope. Roaring must also not be confused with other sounds which are made by horses more in the way of play (grunting, snorting, blowing, etc.).

**Whistling.**—In forensic veterinary science this collective name is given to a series of chronic morbid processes in the respiratory air-passages which produce certain breathing sounds combined with difficulty of respiration (tightness). The most important of these is the left-sided paralysis of the vocal chord just described. But other diseased conditions in the larynx may also produce the so-called laryngeal whistling, especially new growths on the epiglottis or within the larynx (polypi, cysts), chronic laryngitis and peri-laryngitis which provoke stenosis, ossification of the arytenoid cartilage, etc. It is also caused by various chronic diseases of the nasal cavity and wind-pipe, whenever these produce narrowing of these respiratory organs (new growths, stenoses). Diseases also of the guttural pouch, posterior nares or pharyngeal cavity may be followed by whistling.

**Therapeutics.**—The treatment of roaring and whistling is essentially operative. Günther excised the paralysed vocal chord and the sunken arytenoid cartilage after previously opening the laryngeal cavity, and Stockfleth, Fleming and Bassi have followed his method. Möller operates for one-sided paralysis of the laryngeal vocal chord by cutting away the entire arytenoid cartilage. Cadiot and others have adopted Möller's method (arytaenectomy). Other manipulations, such as tracheotomy or the use of devices for compressing the nostrils, merely act as palliatives. Whether a prolonged course of arsenic or iodine (iodide of potassium), or the application of electricity can procure improvement or cure, as many affirm, must be more thoroughly enquired into. In any case such results can only be expected at the beginning of the condition. On the other hand, it is advisable at the outset to try subcutaneous strychnine injections in the vicinity of the larynx ( $\frac{1}{2}$  to  $\frac{1}{4}$  grains of strychnine nitrate per diem); omitting the injection every third day. If it be desirable to apply the

injections intra-tracheally, the dose must be smaller, say, half the above, otherwise signs of poisoning and fatal strychnine tetanus may occur. Veratrine acts similarly to strychnine. As a prophylactic it is absolutely necessary to exclude from breeding all horses with the hereditary taint.

**Spasms of the Glottis** (*spasmus glottidis*), which is the contrary of laryngeal paralysis, is very rarely seen in our domestic animals. It consists in spasmodic closing of the rima glottidis with extreme inspiratory dyspnoea caused by affection of the superior laryngeal nerve. Very little is known as to its causes. Gerlach describes "spasmodic tightness of breath" as a special form of roaring or whistling; but the poisonous agents which he regarded as causing it, viz., chick peas, etc., are among those which produce paralysis of the larynx. Degive and Ebiner have each described a case of true laryngeal spasms in horses. As regards treatment, injections of morphia or chloral hydrate are recommended.

## CHAPTER XXIII.

### DISEASES OF THE TRACHEA AND BRONCHI.

#### ACUTE BRONCHIAL CATARRH. BRONCHITIS CATARRHALIS ACUTA.

**Forms of Bronchitis.**—As with laryngitis, so also in bronchitis we may distinguish various forms according to their duration, intensity and locality. There is also an acute and a chronic bronchitis, a catarrhal and a croupous, a serous, mucous, purulent and foetid. When there is much serous exudation, we speak of bronchorrhœa serosa, or, if specially purulent, of broncho-blennorrhœa. Much importance is also attached to inflammation of the smallest bronchi, the bronchioli, to which the name of bronchiolitis, or bronchitis capillaris, is given. A very rare form is bronchitis villosa. Bronchitis verminosa and mycotica are peculiarly etiological forms. In addition we speak of infectious, glanderous and tuberculous bronchitis.

**Etiology.**—Acute bronchial catarrh is commonest in horses, and next to them in dogs and cattle. The disease is seldom confined exclusively to the mucous membranes of the bronchi, but rather is often found in those of the trachea and larynx, even the nasal cavity being frequently affected. In these latter cases we speak in general of a "catarrh of the air-passages." The causes are numerous.

1. Among *predisposing agencies* are youth, feeble constitution, poor condition of nourishment, pampering and accustoming animals to warm stables.

2. *Cold* is here a main source of the evil, as in laryngitis, especially the action of cold air in breathing. Damp, wet-cold weather, rapid alternations of season, moist, cold winds and thick mist are very prejudicial in this respect, which explains the frequency of the complaint in spring and autumn.

3. *Mechanical and chemical irritants*, e.g., the inhalation of

pungent gases and vapours, hot air, smoke or of air defiled with fungi, as in serving fodder which is dusty, mouldy or infested with uridines or ustilaginee, or the penetration of drugs from carelessly administered drenches. Whether inhalation of pollen from flowering grasses produces bronchitis, as occurs in the hay-fever of man, is uncertain. Respecting parasites as causes of bronchial catarrh, see the paragraph on bronchitis verminosa (further on in this chapter).

4. The disease not unfrequently arises from the spread of catarrh from the upper air-passages to the lower.

5. In a few cases we must presume the action of *infectious matter*, when, for instance, it is sure that cold has not been the cause, or when the malady appears suddenly in several animals at once, with high fever and displaying a strikingly enzoötic and infectious character. Such outbreaks of bronchitis occur sometimes epidemically among horses and cattle, and have been described by several as "epizoötic catarrh of the air-passages."

6. Finally, bronchitis occurs secondarily as an *accompanying symptom* of all kinds of *infectious diseases*, in which it sometimes represents the primary local affection, thus in the distemper of dogs, pleuro-pneumonia, influenza, glanders, anthrax and pox, or variolæ. For this reason it is not always easy to distinguish a secondary infective bronchitis from a primary, as both may be marked by high fever. But in general it is always well, when suspecting a primary and very feverish bronchitis, to think also of the possibility of a secondary affection in the course of some infectious disease, since an inflammation of the lungs, for instance, may at the outset present the symptoms of bronchitis. The former designation of "catarrhal fever" always included such a supposition.

**Pathological Aspect.**—The most important changes on the mucous membrane are redness, swelling and ecchymoses, the deposit of a serous, mucous or purulent secretion caused by transudation of blood-serum, mucous desquamation of epithelium along with breaking down of the mucous glands and emigration of white blood-corpuscles, as well as small cellular infiltration of the mucosa or sub-mucosa. Should the inflammation spread from the mucous membrane to the alveoli, then the parenchyma of the lungs is also affected, and we have a catarrhal inflammation of the lungs (broncho-pneumonia).

**Symptoms.**—Acute bronchial catarrh begins sometimes with moderately high fever (103 to 107° Fahr.). In seventeen such cases among horses we found fifteen times the maximum degree of fever on the first day, the temperature being in four cases over 105.8°, in seven above 104°, and in four exceeding 103° Fahr. The pulse also is quickened, and not unfrequently shivering fits, with great lassitude, are also noticed. Appetite, rumination and milk-secretion are all suppressed. The cough, one of the most important symptoms in acute bronchitis, is at first very dry and painful, but afterwards becomes looser and moister, and is at last accompanied by a watery, glairy, mucous or even purulent discharge. Upon auscultation of the lungs we hear at first rough, intensified vesicular breathing. Later, this becomes indefinite, and we hear dry, rattling noises (whistling, wheezing, buzzing sounds), which grow more moist as the secretion increases. In capillary bronchitis (bronchiolitis) we perceive rustling sounds, especially in dogs. These noises are the characteristic symptoms of bronchitis. In contrast to these serious changes as revealed to the ear, the results of percussion are perfectly normal, so long as there is no development of catarrhal pneumonia. The tightness of breathing is very severe, especially in bronchiolitis, and may even lead to suffocation. Such cases of capillary bronchitis occur mainly in very young or very old animals, in the latter of which the bronchiolitis may very speedily be complicated with oedema of the lungs. According to St. Cyr, the danger in bronchiolitis is in inverse proportion to the girth of sick dogs. The *duration* of acute bronchial catarrh is frequently only four to eight days, but averages two to three weeks. Excepting with very young or very aged animals, its *course* is usually favourable, and the illness ends in complete recovery. But in severe cases, and with continuance of its causes, the acute bronchitis may become chronic.

**Therapeutics.**—In the first place local treatment may be attempted by inhalations of steam, alone or in conjunction with common salt, chloride of ammonium, creolin, carbolic acid, alum, tannin or oil of turpentine. Among internal remedies apomorphine hydrochloride is one of the most valuable expectorants. It is administered to dogs in daily doses of  $\frac{1}{4}$  to  $\frac{1}{2}$  grain, in  $1\frac{1}{2}$  oz. of water, a tablespoonful every three hours. Chloride of ammonium is particularly good for a dry cough, in advanced stages of the catarrh, as well as in slight fever. To horses give it in doses of 2 to 4 drams, and to dogs of 3 to 15 grains. Tartar

emetic is most suitable in higher fever or for more robust animals, and the same applies to yellow sulphide of antimony, a remedy formerly much used (horses 30 to 75 grains; dogs  $\frac{1}{2}$  to 3 grains). Chloride of ammonium and tartar emetic may be prescribed together as a so-called expectorant mixture, e.g., for a dog : ammonium chloride and liquorice juice,  $\frac{1}{2}$  to  $1\frac{1}{2}$  drams; tartar emetic, 8 grains; water, 10 oz.; one tablespoonful three times daily (teaspoonful for small dog). For excessive secretion of the bronchial mucous membranes administer oil of turpentine or terebene (to dogs internally  $1\frac{1}{2}$  to 8 minims), senega-root, aromatic spirit of ammonia, etc. The two latter may be prescribed together thus : infusion of senega,  $2\frac{1}{2}$  drams to 5 oz.; aromatic spirit of ammonia,  $1\frac{1}{2}$  drams. From two teaspoonfuls to two tablespoonfuls daily for a dog. In very painful attacks of coughing we prescribe, as in acute laryngeal catarrh, morphia and bitter-almond water ( $1\frac{1}{2}$  grains to  $2\frac{1}{2}$  drams in 5 oz. of water; daily two to three table or teaspoonfuls for a dog), or codein (3 grains in 5 oz.). Should œdema of the lungs threaten, administer stimulants (camphor, ether, caffeine, hyoscyamine, alcohol), or give an emetic. Alongside this medical treatment there must also be careful supervision of the diet and other hygienic details

#### CHRONIC BRONCHIAL CATARRH. BRONCHITIS CATARRHALIS CHRONICA.

**Etiology.**—There is a natural pre-disposition to chronic bronchial catarrh in all feeble and pampered animals, especially in young dogs and cachectic sheep. Among its direct causes must first be cited those given for acute bronchitis, for the chronic form usually develops from the acute. Then there are certain chronic heart and lung diseases from which it develops secondarily. Among such are valvular defects, chronic interstitial inflammatory processes in the lungs, emphysema of the lungs, all of which cause a regurgitation of blood into the bronchial veins and arteries (the capillary network of the bronchial arteries also communicates with that of the pulmonary artery). On this account most broken-winded horses also suffer from chronic bronchial catarrh. Finally, many general diseases are accompanied by chronic bronchitis, particularly the so-called constitutional diseases, like anaemia, leucæmia, chlorosis, and also chronic Bright's disease. For parasites as a cause

of this complaint, see further on in this chapter under the heading of "Verminous Bronchitis."

**Pathological Aspect.**—In chronic catarrh the bronchial mucous membrane is a dark-brownish red, traversed by greatly dilated vessels. It is thickened and sometimes covered with warty excrescences, the secretions are mucous or purulent, often even putrescent and malodorous (bronchitis foetida), and frequently quite fill the small bronchi, so that the affected lobe of the lung is in a condition of atelectasis, which during further course of the disease is complicated with chronic, catarrhal and interstitial processes in the lungs. When the inflammation is long continued, the bronchial walls become thickened in every part (mucous membrane, the true bronchial tube and the peri-bronchial tissue) by new growths of connective tissue after previous small-cellular infiltration: endo-bronchitis, meso-bronchitis and peri-bronchitis chronic. A special form of peri-bronchitis in horses has been described by Dieckerhoff as peri-bronchitis nodosa. This appears as multiple, tough nodules, greyish-white in section and varying in size from a groat or millet-seed up to a pea; the interior is often filled with caseous and calcified bronchial secretion, so that the nodules greatly resemble the tubercles of glanders. But they are distinguishable from the latter by their regular quality and equality of age and by the fact that their centre can be peeled or decorticated—a fact denied, however, by Nocard. According to Ziegler, peri-bronchitis nodosa should rather be reckoned as a pneumonia, so far as it concerns the breathing bronchioli, and be designated as nodular broncho-pneumonic induration.

One of the most frequent sequels of chronic bronchial catarrh is bronchiectasis, i.e., dilatation of the bronchial walls, which, being catarrhally inflamed, are therefore less resistant; this occurs especially in horses at the apex of the lungs. We distinguish a cylindrical, a sacculated and a spindle-shaped bronchiectasis. The bronchiectatic cavity is fully charged with decomposed, thickened mucus and pus, which may even become calcified (so-called "lung-stones," or pulmonary concretions) and attain the size of a pea or chestnut, or even that of the fist. Smaller bronchi are often distended to the thickness of a finger. Finally, another not infrequent accompaniment of chronic bronchial catarrh is emphysema.

**Symptoms.**—These are in general the same as those of

acute bronchitis. Here, also, the cough is the chief symptom, and it is usually combined with profuse secretion of mucus. Auscultation reveals various rattling noises, partly dry and partly moist. The difficulty of breathing is extremely acute whenever the chronic bronchial catarrh is complicated with atelectasis and emphysema. In the horse we then have the phenomena of broken wind. As the disease progresses the animal's condition begins to suffer; it grows thin, feeble and cachectic and sometimes perishes of catarrhal pneumonia. The presence of bronchiectasis is known by the enormous and sudden discharge of sputum, which is usually much decomposed and smells very badly, and also—though but rarely—by a localised, clear tympanitic tone on percussion of the thoracic wall.

**Therapeutics.**—A cure of chronic bronchial catarrh is often possible, but takes a long time. The remedies are the same as those advised for the acute form. Expectorants, which may be administered both internally and also externally as inhalations, are most frequently used, such as oil of turpentine, tar, creolin, chloride of ammonium, etc. Also the neutral salts, apomorphine, ipecacuanha, senega and quillaja are in use. The intratracheal injections recommended by Levi and others have been already tried by Lafosse 50 years since, and their efficacy is doubtful. In man the treatment of bronchiectasis is partly surgical.

**Physetigmine and pilocarpine** have latterly been recommended for chronic bronchitis. But we must counsel caution in their use, for in one case, after subcutaneous injection of  $1\frac{1}{2}$  grains of the former for a powerful horse suffering from chronic bronchial catarrh, we observed extremely difficult breathing, followed after three-quarters of an hour by death from suffocation as a result of oedema of the lungs. In a similar manner we have seen dogs die from oedema of the lungs after being treated for the same disease with  $\frac{1}{2}$  grain of pilocarpine.

#### BRONCHIAL CROUP. BRONCHITIS CROUPOSA.

**Etiology.**—This arises from the same causes as laryngeal croup. But in cattle there seems to be in addition a specific, infectious bronchial croup. We also meet with the disease as a co-symptom of croupous inflammation of the lungs, also in many infectious diseases, e.g., in rinderpest, malignant catarrhal fever and bovine pleuro-pneumonia.

**Pathological Aspect.**—The mucous membranes of the

trachea and bronchi are injected and swollen, showing yellowish membranous coatings up to  $\frac{1}{4}$  inch thick, which possess a smooth upper surface and adhere only loosely to the mucous membrane. Its microscopical constitution is the same as in laryngeal croup. Sometimes we also find rolled-up, ribbon-like masses, especially on the posterior wall of the wind-pipe. Moreover, the finer bronchi are often completely choked with yellow, tough cylinders like thrombi. The lung is very hyperæmic, and for the rest we have all the familiar signs of suffocation.

**Symptoms.**—In a few cases the sickness begins with certain general phenomena, such as lassitude, poor appetite and slight cough; but most commonly it appears suddenly and with severe derangement. The beginning is usually a tightness of breath amounting to asphyxia. Breathing is quickened and is often rattling, snoring and gasping. The cough is often persistent and painful; and during its attacks ribbon-shaped or tubular, croupy matter is expectorated, even as much as half a yard in length. Upon auscultation of the trachea and bronchi, we can hear gurgling, bubbling, quivering and rattling murmurs, and can feel tracheal fremitus with the hand. Percussion of the thorax is normal. In addition we observe signs of dyspnoea and asphyxia, viz., cyanosis of the visible mucous membranes, breathing with widely-opened mouth and outstretched tongue, anxious look, etc. The *duration* of the attack is brief, and it has usually attained its height by the second or third day and ends speedily, either with ejection of the croupy matter or with death from suffocation. In youthful animals with a small trachea the latter is the usual conclusion. The total length of the disease does not exceed six to eight days. Cattle and sheep are most liable to its attacks.

**Therapeutics.**—Owing to its very rapid course the treatment of bronchial croup is usually unavailing, the more so as even tracheotomy can do little or no good. The utmost that can be done is to try and loosen the croupy membrane by inhalation of steam or vapour of lime-water, while to smaller animals one may also give an emetic. But in general any treatment usually comes too late, and with all severe cases in cattle early slaughtering is the wisest step. According to Trasbôt, blood-letting sometimes produces rapid improvement.

## VERMINOUS BRONCHITIS. LUNG-WORM SICKNESS. HUSK. HOOSE.

**Etiology.**—Lung-worm sickness, or lung-worm plague, also known as lung-worm cough, is caused by animal parasites of the nematode or thread-worm order and by some species of *strongylus* (palisade-worms), which penetrate to the trachea and bronchi and produce at first a verminous bronchitis and catarrhal pneumonia, but later a verminous inflammation of the lungs. In our several domestic animals the following strongyli are of clinical importance :

1. In **sheep** (and goats) chiefly *strongylus filaria*. Less common is *strongylus paradoxus* and *str. rufescens* (*commutatus*). Besides these we must mention the hair-worm, called by Koch "*pseudalius ovis pulmonalis*," and by Müller "*pseudalius capillaris*."

2. In **cattle** *strongylus micrurus* (very rare also in horses and asses).

3. In **pigs** (and in man) *strongylus paradoxus*.

4. In **dogs, cats and rabbits** various nematodes resembling *strongylus*, but not yet more exactly known.

5. In **poultry** the *syngamus trachealis*, which belongs to the strongyli. (See diseases of the breathing organs in birds).

Besides these there occurs in **hares** a localised verminous pneumonia with caseation, which resembles tuberculosis and is caused by *strongylus commutatus*.

**Zoological Notes.**—The lung-worms which occur in man are also extremely diffused as parasites throughout the animal world. The following mammals are thus infested, in addition to those named above :

1. *Goat and camel* : *strongylus filaria*.
2. *Dog* : *strongylus canis bronchialis* (Osler).
3. *Chamois and antelope* : *strongylus filaria* ; *pseudalius capillaris*.
4. *Cat, hare, and rabbit* : *strongylus commutatus*.
5. *Fox* : *cremocoma semi-armatum* ; *trichosoma aërophilum*.
6. *Marten, polecat and weasel* : *filaroides mustelarum*.
7. *Dolphin* : *pseudalius tumidus*, *convolutus*, *minor* and *reflexus*.
8. *Hedgehog* : *trichosoma tenue* ; *crenosoma striatum*.

We also meet with lung-nematodes in numerous *birds*, *reptiles* and *amphibia* (frog). Thus *syngamus primitivus* is found in the domestic fowl, pheasant, turkey, duck, peacock, partridge, magpie, crow, swallow,

starling and woodpecker; *syngamus trachealis* in the domestic fowl, pheasant, partridge, stork, etc. Müller has compiled a detailed medical and zoological treatise on all the lung-worms at present known in the various animal species. According to his report, *strongylus rufescens* and str. *commutatus* are identical.

**Natural History Notes.**—In its sexually ripe state the palisade-worm inhabits the air tubes and bronchi of sheep more particularly, and after them of cattle and pigs. It is there that the eggs and embryos are produced. The thread-worms, along with their eggs and embryos, are coughed up by their hosts, when the embryos complete their development in the open, probably in pools and swamps. Whether they use any intermediate host, such as insects, rain-worms or snails, for this purpose or not, is not surely known, but it is likely. The absorption of the worm-brood into the bodies of their hosts occurs mostly through drinking-water or fodder. But it is possible that the brood reaches their air-passages through the inhalation of dried mud.

Infection usually takes place in spring, for after harvest, according to Gerlach, it no longer occurs. But Tapken's observations prove that there are exceptions to this rule; for in the marshes along the Weser and Jade the immigration of the brood into the cattle takes place chiefly in July and August. The *strongylus micrurus* requires there six to seven weeks for development, and, when the disease runs acutely, the animals—chiefly calves—may die in a further six or seven weeks. But in sheep infection usually occurs in spring. The disease makes its appearance in autumn, and attains its greatest spread in wet seasons, whereas in dry ones it abates and may quite disappear for a time. Thus on one of the Alps, Bauer saw it return after eight years, the summer being a very wet one. We see then how important water is for the development of the worm-brood. In certain damp, swampy and peaty spots lung-worm sickness is constantly present as an enzootic malady, but has never yet been noticed where animals are stall-fed. The infection of one animal by another cannot be accepted. It is probable that the brood first attain the stomach, passing thence through the paunch into the pharyngeal cavity (rumination in sheep and cattle), and through the trachea to the bronchi. There is no evidence for the assumption that they reach the lungs through the blood-stream. In the bronchi the *strongyli* can complete their full development. But very frequently the young *strongyli* advance from the bronchi into the pul-

monary tissue as far as the pleura, where they often settle in large cavities (worm-nodules). Thence they wander later back again to the bronchi, where they attain sexual maturity and full size and often accumulate in large quantities, thus producing bronchitis and bronchiectasis, as well as congestion of the larger bronchial tubes, with occasional sudden death from suffocation. From the bronchi they are then coughed out also with their eggs and embryos, and the circle of their existence is thus completed.

In contrast to this emigration of the pulmonary thread-worms (*strongyli*) from the lungs, the pulmonary hair-worms (*pseudalium capillaris*) remain behind in the lung-tissues, where they become encapsuled and die.

The vitality of palisade-worms and their embryos is very great. Thus Colin saw embryos which issued from the bursting bodies of sexually ripe *strongyli* and remained alive in water for over two months. Ercolani reports that the parasites were not destroyed after being dried for 30 days and then subjected for eight days to the action of spirit or solution of sublimate. Railliet states that the young embryos are capable of development after being dried for eight months.

**Occurrence.**—As a complaint attacking flocks, verminous bronchitis works great havoc sometimes among sheep. Carnet reports that on the frontiers of Morocco half the entire sheep and one-third of all cattle have been known to fall before the plague. In 1883-86 several million sheep, three-fourths of all flocks, perished of this disease in Buenos-Aires. It occurs much more rarely in enzoötic form among cattle. But sporadically we very often find lung-worms in domestic animals without any general injury being caused. In Berlin lung-worm sickness is found as a local lung-affection in 8 per thousand of all pigs slaughtered and 3 per thousand of all sheep; but in such cases only the lungs are condemned by the meat-inspectors. Owing to the malignancy of the disease, it is regarded in some countries as one against which legal warranty can be demanded, the term in Austria being two months.

**Pathological Aspect.**—The changes effected by *strongyli* in the bronchi and lung-tissues are of very varied nature. Sometimes they are the phenomena of chronic bronchitis with bronchiectasis, or at others we observe lobular, catarrhal-pneumonic foci, or perhaps new growths like nodules,

the so-called pseudo-tuberculous pneumonia or nematode-tuberculosis. In goats, according to Bugnion, even diffuse pneumonia occurs, caused by the eggs and embryos of palisade-worms.

1. *Verminous bronchitis* consists in much swelling, as well as suppurative and haemorrhagic inflammation of the bronchial mucous membrane, in which the embryos are not rarely found enveloped in clusters. The trachea and bronchi are puffed out like bags where the worms have lodged, and they are found there rolled up into balls and surrounded by mucus and pus. The lumen of the bronchi is full of a tough, purulent and not seldom bloody mucus, in which both eggs and very active embryos are found. The lung-tissue round about the bronchiectasis is thickened, atelectatic, splenetic and partly also emphysematous; the lungs themselves being oedematous and devoid of blood.

2. The *lobular pneumonic foci* may exist along with the verminous bronchitis just described. They develop as a result of the inflammation spreading to the lung-tissue in the form of circumscribed hepatized areas, which during further progress of the disease may change into atelectasis, splenization or formation of abscesses. These changes are found especially in calves and pigs, and starting from clusters of mature strongyli. In pigs these thread-worms often occur only in the apexes and on the edges of the lungs and, according to Kohlhepp, we may suspect their presence from superficial examination, whenever the apexes and edges of the lung are somewhat thicker, or if some of its lobes do not fall together sufficiently. Sometimes we also find slight, local pleuritic changes.

3. The *pseudo-tubercular pneumonic foci* represent tubercle-like nodules of different sizes, with which the lung-tissue is often completely permeated, and which may be felt through the tissue near the sharp edge of the lungs. Not unfrequently they produce quite a lumpy surface on the lungs and even pleuritic adhesions. These nodules are simply nests of worms with a connective-tissue capsule, from which, on cutting them, plugs can be pressed consisting of pus and *Strongylus*-embryos. They are small peri-bronchitic foci of inflammation proceeding from the bronchi.

In addition to the numerous changes in the lungs we find, in advanced cases, symptoms of hydramia, effusion of water into the body cavities and connective tissue, as well as general anaemia. In pigs we find more especially gelatinous swelling

of the mesenteric, peri-laryngeal and retro-pharyngeal connective tissue.

**Lung Hair-worm Sickness.**—Koch contrasts the lung thread-worm sickness just described with that caused by the hair-worm which he calls *pseudalium ovis pulmonalis* (according to Railliet this worm is identical with *Str. Rufescens*). This parasite, which also belongs to the nematodes, was seen by Brown, Cobbold and Axe as long ago as 1851, and was named "*nematoideum ovis pulmonale*" by Gray Sandic and Padley. Later investigations by Müller have shown that the hair-worm also occurs in the chamois. He therefore proposes for it the more general name of *pseudalium capillaris*.

The history of the hair-worm's development is at present not fully known, but it is probably like that of the thread-worm. Infection in any case takes place through food and drink. The worms pass from the stomach (rumination) through the pharyngeal cavity to the trachea and bronchi, whence they invariably penetrate to the lung-tissue and there become encapsulated. Only the larvæ, and not the original worms, escape from the bronchi to the open air (Müller). These hair-worms are fairly plentiful. According to Ranke, almost all the sheep brought to London markets are infested by the worm. Ruser found it in 19½ per cent. of all slaughtered sheep, and Motz in 4.2 per cent., and that pretty evenly all the year round, the *pseudalium* being in the lungs. The pathological changes in the lungs are very varied, and even with the naked eye two distinct forms of nodule, or pseudo-tuberculous pneumonic focus, can be distinguished.

1. Small, whitish-yellow nodules, the size of miliary tubercles, which on section are found to be solid and partly calcified and containing very small worms visible by the unassisted eye. Under the microscope they reveal an accumulation of white blood-corpuscles and in the centre a number of hair-like round worms rolled up into a ball. On the periphery of these smaller foci of inflammation is a capsule of connective tissue.

2. Larger, sharply-defined knobs, of yellow to yellowish-grey colour and from a lentil to a walnut in size, which are some of them elastically soft and others firm and hard. Their section shows small hair-like worms which are partly brown (males) and partly white (females) amid a milk-white, frothy fluid. The larger nodules only contain caseous matter.

The symptoms, according to Koch, are cough and increased secretion of mucus, but there is no expectoration of worms. Where the number of these is only small, no morbid signs are perceptible; but severer cases exhibit the symptoms of pulmonary phthisis and cachexia. According to Ströse, the parasite occasions no disorder in older animals, only in lambs is there cough, increased mucous secretion, emaciation and, in a few cases, periodical nose-bleeding. But the lambs also mostly recover. Prognosis is therefore favourable, especially for older animals. Diagnosis during life is based on proof of the eggs and embryos in the sputum.

**Symptoms.**—1. In sheep the signs of lung-worm sickness are those of severe chronic bronchial-catarrh with subsequent pulmonary phthisis. Of all domestic animals sheep sicken most frequently and most severely, lambs and yearlings

being especially attacked. They begin with slight cough, which soon grows stronger, croaking and gasping, but at last is very weak and faint. This cough is noticed most when in movement, so that when driving a flock it seems as though they are all coughing. There is at the same time a mucous sputum, with salivation, slavering and choking, and sometimes entire lumps of worms are coughed up, when the sheep often rub their noses on the ground. Breathing becomes quicker, is rattling and gasping, and upon auscultation of the air-tubes and bronchi the most varied rattling sounds can be heard. These are complicated with a thin, mucous nasal discharge. As the ailment progresses breathing grows more and more difficult; the animals are very faint, eating, for instance, upon their knees; skin and mucous membranes become paler; the quality of the wool deteriorates daily; and death follows after weeks or months from exhaustion, unless suffocation put an earlier termination to the malady. The ratio of mortality varies with the age of the animals, their condition and feeding from 10 to 70 per cent.

2. In cattle the symptoms are quite similar, and here, too, calves and young animals from one to one and a half years of age are most frequently affected. The ailment begins with attacks of strong coughing; appetite and general condition being at first undisturbed. The course varies according to age and degree of infection. Calves may sometimes die of suffocation during the first week of sickness, but the condition generally lasts for several weeks. In most cases, with proper dietetic treatment, recovery takes place spontaneously. But sometimes a growing weakness and emaciation are developed as a result of chronic lung affection, when the cough grows fainter and breathing more rapid. In such cases the symptoms of verminous bronchitis awaken suspicions of bovine pleuropneumonia or tuberculosis. But in general cattle have more power of resistance, and the mortality is less with them than among sheep. During an epidemic outbreak arising from *Strongylus micrurus*, Claes has lately observed in almost all the sick cattle ophthalmia caused by *Filaria papillosa* in the anterior chamber of the eye. Stall-cattle generally escape the disease.

3. Among pigs *Strongylus paradoxus* does not seem to be at all rare in the lungs, but these animals seem to be much less sensitive to round worms than cattle. Certainly

full-grown pigs very seldom suffer. But the disease is fairly common among young pigs, appearing even enzootically. But one notices that different individuals suffer in very different degrees, and generally the malady abates as the animal grows. Sudden death from suffocation and oedema of the lungs occurs sometimes, though not so often as has been supposed. In very young animals chronic debility is often developed.

**Strongyli in other Animals.**—Here they are much more rarely found. Laulané discovered tubercle-like nodules ("pseudo-tuberculosis") in the lungs of a dog caused by irritation from the eggs of *Strongylus vasorum*, which had matured in the right ventricle and pulmonary artery, whence the embryos emigrated to the bronchioli. In a cachectic dog which died of ascites Railliet and Cadiot found severe strongylosis of the heart and lungs (numerous embryos of str. *vasorum* in the bronchial secretion and the trachea). Osler and others also found a round worm like a filaria in the air-tubes and bronchi of dogs, which produced warty nodules upon the mucous membrane of the trachea and bronchi (tracheitis verrucosa), and were from a millet-seed to a coffee-bean in size. They produced an epizootic outbreak like distemper.

**Therapeutics.**—In lung-worm sickness prophylaxis consists in the destruction of all infected lungs and in the avoidance, if possible, of swampy pastures in spring, a measure which unfortunately cannot always be adopted. Against the disease itself no drugs or treatment of certain effect are at present known. It has long been the custom to use fumigations and inhalations of tar, carbolic acid, creolin, oil of turpentine, tobacco, etc., the animals being shut up for some time in a closed space filled with the vapour in question. These vapours are supposed to make the animals cough up the bronchial mucus and the worms it contains. At the same time steps must be taken to secure the destruction of expectorated worms. For pigs an emetic is recommended (veratrine  $\frac{1}{2}$  grain). More important though is good, substantial nourishment and the administration of tonics, especially preparations of iron, in order to keep up strength. Of late years several practitioners have recommended the intra-tracheal injection of anti-parasitic remedies, especially carbolic acid and oil of turpentine, also of creolin, chloroform and oil of cloves. Eloire used a mixture of olive oil 3 oz., oil of turpentine 3 oz., carbolic acid 30 grains, stinking animal-oil 30 grains, of which for three days he injected  $2\frac{1}{2}$  drams daily. A mixture of oil and oil of turpentine 3 oz. of each, with  $1\frac{1}{2}$  drams of creolin, may be used in the same way. But Dieckerhoff, Tapken and others were unable to report undoubted benefit from these injections.

## CHAPTER XXIV.

### DISEASES OF THE LUNGS.

#### INFLAMMATION OF THE LUNGS. PNEUMONIA.

**General Remarks on the Sub-division of Lung Inflammation.**—In no other organ of the body do so many varieties of inflammation occur, both as regards character, seat and extent, as well as cause and course, as in the lungs.

1. According to the *pathological nature* of the exudate we may divide lung-inflammation into croupous or fibrinous, catarrhal, haemorrhagic, suppurative, necrotic, caseous and desquamative ; and may also consider inflammatory oedema as a serous form.
2. According to its *seat and extent* we distinguish between lobar, lobular and miliary pneumonia, as the larger, smaller or smallest areas of the lungs may be attacked ; also interstitial and inter-lobular pneumonia, the former being marked by emigration of white blood-corpuscles into the interstitial connective tissue with consequent proliferation of this tissue, and the latter by special affection of the inter-lobular lymph-vessels, whereby, as in bovine pleuro-pneumonia, the parenchyma of the lungs is isolated, as it were, by thickened and fully distended lymph-spaces, whence this form also bears the name of dissecting pneumonia. Finally, we distinguish a broncho-pneumonia and a pleuro-pneumonia.
3. From the etiological standpoint we also speak of a genuine, i.e., a primitive idiopathic and independent pneumonia ; and furthermore of infectious, glanderous, tuberculous, actinomycotic, mycotic, verminous and traumatic inflammation of the lungs ; of an aspiration, an inspiration and a swallowing (due to foreign bodies) pneumonia ; of haemogenous, metastatic, hypostatic and pleurogenous forms. Among infectious diseases during which pneumonia is developed we must name equine and bovine pleuro-pneumonia, swine-fever, distemper of dogs, influenza, pyæmia and septicæmia. Vagus-

pneumonia is an aspiration pneumonia, which is artificially produced by section of the vagus. 4. In accordance with its course we may subdivide inflammation of the lungs into benign and malignant, abortive, ephemeral (lasting one day), fixed, i.e., confined to the spot first attacked, wandering and erratic, i.e., inconstantly wandering and shifting its seat; names which all point to an analogy between pneumonia and erysipelas. Malignant pneumonia with extreme debility, was formerly called typhoid; and bilious when accompanied by icterus caused partly by decomposition of blood and partly by swelling of the liver and duodenal catarrh.

In all the various species of animals we find various forms of inflammation of the lungs. In *horses* the chief form is contagious pleuro-pneumonia (murrain), and also the primary croupous and the catarrhal pneumonia; in *dogs* the catarrhal pneumonia of distemper; in *cattle* the tuberculous and interlobular pneumonia of bovine pleuro-pneumonia; in *pigs* the necrotic pneumonia of swine-fever; and in *sheep* verminous pneumonia.

Naturally among such a number of forms each one cannot be separately discussed. For the purposes of clinical practice we shall therefore confine ourselves to a description of the following forms:

1. Croupous (genuine, primary) pneumonia in horses, cattle and other domestic animals.
2. Catarrhal pneumonia.
3. Pneumonia due to foreign bodies.
4. Mycotic pneumonia.
5. Interstitial pneumonia.
6. Metastatic pneumonia.

#### I.—CROUPOUS (GENUINE, PRIMARY) PNEUMONIA.

##### (A). CROUPOUS, SPORADIC PNEUMONIA OF THE HORSE.

**Etiology.**—During recent years the question has been frequently raised whether there be a croupous pneumonia of horses apart from murrain (infectious pleuro-pneumonia)? On this question we hold the affirmative opinion, as hitherto no sure proof of etiological connection between the two has been adduced. We have ourselves no doubt that, although the majority of pneumonia cases in horses belong to murrain, yet there is an inflammation of the lungs appearing sporadically

in horses, and which is quite independent of murrain. Many other observers have adopted this our view on the ground of their practical experience. Primary croupous pneumonia of horses is analogous to the sporadic croupous pneumonia of cattle, which also is independent of bovine pleuro-pneumonia. Of known external causes two must be specially mentioned, viz., over-exertion and cold. Inflammation of the lungs evidently develops from both of these in connection with a previous active, hyperæmia of the lungs. As other causes we may add : breathing hot air or irritant vapours (smoke, chlorine, sulphurous acids).

**Pathological Aspect.**—In general, croupous pneumonia is characterised by inflammation of the alveoli and bronchioles with formation within the alveoli of an exudate rich in fibrin which quickly hardens, whereby the parenchyma of the lungs usually remains intact after the inflammation has passed away. Croupous pneumonia is, moreover, generally lobular, i.e., spread over larger divisions of the lungs, and it is the anterior lower part which is most frequently attacked. In a majority of cases the inflammation is on one side only. The pathological aspect varies with the different stages. According to custom we distinguish the following :

1. *The stage of congestion (engorgement).* This lasts about one day. The lungs are in a condition of extreme congestive hyperæmia, intensely reddened and swollen, retain the impress of the fingers, crackle but slightly when cut and present a smooth, moistly lustrous section-surface, marked injection of the capillaries and even haemorrhages. The lumen of the alveoli or bronchioli gradually fills with a serous fluid, rich in albumen (inflammatory oedema of the alveoli), with which red blood-corpuscles are mingled, due to the haemorrhages, and also white ones which have penetrated thither, and likewise large quantities of desquamated lung-epithelium. The interstitial tissue is gelatinous and infiltrated with oedematous fluid, and the bronchi are charged with a fine, frothy fluid. The air is thus driven out of the alveoli and bronchioli and partly also from the bronchi.

2. *The stage of red hepatization.* This begins with coagulation of the fibrinous exudation and lasts on an average two days. As the fibrinogenous substance of the blood-plasma and the fibrino-plastic matter of the white blood-corpuscles combine to form fibrin, microscopically fine threads or nets of fibrin

are produced in the stiffening exudate. The lung is thereby made heavier, sinks in water, appears swollen and enlarged, has a consistency like liver or india-rubber, and is tender and friable; but sometimes it is flabby and so soft that it almost liquefies. The section-surface is greyish-red and sometimes dark red, owing to more intense inflammation and consequent haemorrhage, that is to say, there are darker patches surrounded by greyish-red hepatized tissue (appearance resembling granite): in very rare cases the section-surface is uniformly red. When there is simultaneous hypostasis, this surface is steel-blue, of spleen-like consistence and colour (splenization). In addition the section-surface appears on darker red places to be granular, an effect caused by the fibrinous plugs in the alveoli: these granules, however, cannot be squeezed out.

3. The *stage of yellow hepatization* is produced by dis-coloration of the exudate, breaking down and fatty degeneration of the cellular and fibrous elements, their dissolution and incipient resorption, as well as by emigration of white blood-corpuscles from the capillaries of the alveoli. In this stage the granules upon the section-surface can be removed by pressure.

4. The *stage of resolution* is distinguished by the final liquefaction of all decomposed exuded matter and its resorption or expectoration, the lungs being restored to their original integrity as soon as this process is completed.

Round about the inflammatory foci the lungs are often oedematous and emphysematous. The pleura above any superficially situated pneumonic area is dull, rough and opaque, and occasionally also covered with haemorrhages (*pleuritis sicca*). Fluid exudation in general occurs more rarely. The bronchial mucous membrane is inflamed and has sometimes a croupy coating. The lymphatic glands of the lungs are oedematosly swollen, limp and often soft and pappy; not unfrequently they also are infiltrated with haemorrhages. The mucous membrane of stomach and bowels is often found in a state of catarrh. Liver, kidneys and heart may be in a state of fatty-degeneration. In exceptional cases, and contrary to the usual course, we find necrosis and abscess-formation, and also, in chronic cases, induration and atelectasis of the lung-tissue.

**Symptoms.**—The aspect of primary croupous pneumonia is very similar to that of contagious pleuro-pneumonia. It generally opens with a very severe attack of fever, with

temperature from 105°-107° Fahr. The fever is, during the first six days, usually continuous with only slight remissions, upon which there commonly follows a very rapid fall. The speed of the pulse varies according to the animal's constitution, fluctuating between 60 and 80, or even 100. Very often the fever begins with a long shivering fit, and is manifested by great faintness, muscular weakness, dulness of the sensorium and very red mucous membranes; appetite is suppressed but never quite lost, and evacuation is retarded. In severe cases there is also a jaundiced discoloration of the conjunctiva.

In accordance with the height of the fever and extent of the lung-inflammation, the breathing is both quickened and impeded, counting on an average 20 to 60 respirations per minute. The type is preponderatingly costal, the nostrils being widely opened. The exhaled air is higher in temperature. The animals either do not lie down at all or else only upon the affected side, but rather stand with wide-spread fore legs, timidly avoiding every movement. At the same time a superficial, short and painful cough is observable, which, however, is not seldom entirely suppressed. Also we sometimes see a reddish-yellow nasal discharge.

Percussion of the thorax reveals during the initial stage a tympanitic tone, which is seldom quite clear, but mostly somewhat dulled. The second or hepatization stage is marked by a dull percussion-sound with much resistance under the hammer, but on the borders of the dull resonance we obtain tympanitic sounds. The third stage produces tympanitic tones once more. This typical course of percussion fails in abortive cases only, in which, owing to the very short duration of the ailment, percussion discovers no changes or only quite trifling ones.

Auscultation of the lungs discovers at the outset a rustling or crepitating sound; in the second stage a bronchial breathing, particularly during expiration; and in the third a moist rattle. Auscultation of the healthy lung often gives a stronger vesicular breathing sound, which is also frequently rough. When the pleura also is implicated we hear friction sounds.

In many patients the urine is often alkaline for a long time together; but during the later course is frequently acid. On the other hand, it is usually albuminous, especially during the middle stages of the disease. Its specific gravity is somewhat high, but its chlorides are diminished (exudation into the lungs), while those of the urea are increased.

**Course and Prognosis.**—The usual course of croupous pneumonia is typical : congestion, hepatization and resolution following each other at regular intervals. The malady reaches its height on an average on its fifth or sixth day ; from that point renovation and resorption of exuded matter begin ; the dulness clears sometimes in a remarkably short time, even in 24 hours, but usually by degrees ; we hear rattling and rustling sounds, the cough becomes looser, appetite improves, so that after some 8 to 14 days recovery is complete. But in other and not exactly rare cases, the disease does not run all its stages, but ceases in the stage of congestion or at the beginning of hepatization. This abortive course causes the croupous pneumonia to last sometimes only one, two or three days (ephemeral pneumonia).

**Death by asphyxia** occurs from development of general oedema of the lungs and especially in severe or so-called typhoid cases with general dissolution of the blood, or when the inflammation is wide-spread and accompanied by extreme weakness of heart. The course becomes *chronic* in very old and very young animals, in such as are feeble, and after previous bronchitis and bronchiectasis. In these cases chronic induration of the lungs with new formation of connective tissue may be developed, as well as permanent dulness and dyspnoea ; or suppuration with persistent fever, sometimes also with shivering fits and increase of sputum ; or finally, a necrosis of the lung-tissue with subsequent sepsis and foul smelling breath. In such chronic cases recovery either occurs in six to eight weeks, or a chronic pulmonary phthisis (broken wind) is set up, or else the case ends in death from sepsis.

The *prognosis* of croupy pneumonia is nevertheless on the whole very favourable. The percentage of mortality is very small.

**Differential Diagnosis.**—Primary croupous pneumonia of horses is distinguishable first of all from catarrhal pneumonia by its clinical course. Whereas the former is preceded by active hyperæmia of the lungs, the latter develops from a previous bronchitis. The course of catarrhal inflammation of the lungs is also irregular and ends very frequently in atelectasis and induration ; the inflammation-foci are thereby lobular, so that percussion usually shows only small and localised dulness.

To distinguish croupous pneumonia from pleuritis one must attend especially to the typical course of the pneumonia and the

results of physical examination of the lungs. In pleuritis the line of dulness is mainly horizontal, because the pleuritic exudation, like all liquids, floats upwards, while in pneumonia this line is irregular, rising and falling. Moreover, the percussion-tone in pleuritis is much duller, the bronchial breathing sound is generally absent, and finally, the friction sounds are characteristic of pleuritis. Then also the heart-beat is strengthened in pneumonia, but in pleuritis may be weakened by pressure of the exudate.

General croupous pneumonia is distinguished from contagious pleuro-pneumonia chiefly by its non-contagiousness, and also by the fact that it sometimes attacks horses which have already had the latter disease.

**Therapeutics.**—As a disease of typical and benign course croupous pneumonia demands really no special treatment. The chief thing is dietetic care. But besides this, a diligent application of cold-water bandages about the thorax, renewed at least every ten minutes, has a favourable effect upon the local inflammation. Only when there is a departure from the typical course need one administer any medicines. Thus when there is excessively high temperature, particularly if dealing with animals deficient in resistant strength, one may use febrifuges, and where there is great heart-weakness, digitalis may be tried. We give the latter in daily doses of 2 to  $2\frac{1}{2}$  drams of the powdered leaves, made into an electuary with neutral salts. Among fever-remedies chinin and alcohol are not very effective, whereas anti-febrine and anti-pyrine, in repeated doses of  $\frac{1}{2}$  to  $\frac{3}{4}$  oz. or more, may be cited as very efficient. From its cheapness, anti-febrine is especially useful as a fever-remedy for horses, in two or three doses of 1 oz. In cases of delayed resorption, inhalations and Priessnitz' compresses are used, with internal administration of alkalis, viz., acetate of potash made into an electuary with powdered juniper berries. In gangrene of the lungs inhalations of tar or creolin are advisable. Finally, one may rub the animals vigorously, inject cold infusions into the rectum and also apply an inunction of 6 to 8 per cent. mustard-spirits on the lateral wall of the breast, as an anti-febrile and derivative remedy. For old and very much enfeebled animals alcohol in doses of 3 to 6 oz. given with their drinking-water has done good service from an economical point of view; but even better, if it be available, is wine in quantities of from  $\frac{1}{2}$  pint to  $1\frac{1}{2}$  pints. Trasbôt

recommends milk in considerable quantities (up to 3 gallons a day).

(B).—*CROUPOUS, NON-CONTAGIOUS PNEUMONIA IN CATTLE.*

**Remarks on the Relations between this Complaint and the Contagious Pleuro-Pneumonia of Cattle.**—The existence of a croupous, non-contagious sporadic and acute pneumonia in cattle independent of bovine pleuro-pneumonia has long been stoutly contested, especially by Gerlach and Haubner. The former, in his work on forensic veterinary science, published in 1872, referred every case of independent pneumonia with marbled hepatization to bovine pleuro-pneumonia. And yet the existence of such an independent and non-contagious pneumonia admits of no doubt. Whatever the causes of the many cases reported, and although bovine pleuro-pneumonia may occasionally run sporadically and acutely, yet there remain such a number of exactly observed and carefully described cases, that the occurrence of a primary pneumonia in cattle along with bovine pleuro-pneumonia has been abundantly proved. Besides this, no trustworthy evidence of an etiological connection between sporadic pneumonia and that of bovine pleuro-pneumonia has yet been adduced.

Little or nothing is yet known of the causes of idiopathic croupous pneumonia. Probably they are physical or chemical irritants which reach the lungs with the breath, and it is possible that we have to do with an infectious but not contagious disease. More exact researches on these points are needed.

**Pathological Aspect.**—The changes here are exactly the same as in the corresponding disease of horses. A relative difference is only caused by the different structure of the lungs of the ox, in which the inter-lobular lymph-spaces are wider and are therefore secondarily affected more readily, and which also perhaps permits the inflammatory process to spread more quickly from the lungs to the pleura. As in horses, the inflammation is mainly lobular, and the four stages there described are also here characteristically present. The colour, however, of the section-surface during the stage of hepatization is not so light, but rather dark-brown to dark-red. The inter-lobular connective tissue also generally participates secondarily in the inflammation, being either oedematosely infiltrated or even, though only to a very slight degree, permeated by

fibrinous infiltrate, only a few white blood-corpuscles are however lodged in the fibrin. On the other hand, the pleura is more severely affected than in horses ; along with pleuritis sicca we not unfrequently find the forms of pleuritis serosa and sero-fibrinosa.

**Symptoms.**—These are likewise essentially the same as those described in horses. As a rule the malady makes its appearance suddenly and sporadically. High infective fever, severe general symptoms, great dyspnoea, much groaning and coughing are its most important phenomena. Percussion reveals at first normal, and later tympanitic and muffled tones, and on auscultation we perceive at first crepitus, then bronchial breathing and at last rattling sounds. Croupous pneumonia in cattle is, however, often distinguished from that of horses by its more rapid and unfavourable course. Not unfrequently the ailment ends in the stage of hepatization on the third or fourth day.

**Differential Diagnosis.**—The most important question in croupous pneumonia of cattle is its differentiation from bovine pleuro-pneumonia. The following points will help in so-doing :

1. Croupous pneumonia seems to occur only *sporadically* and often in byres where bovine pleuro-pneumonia has not prevailed for years and tens of years, and where it has never subsequently been seen:

2. The *clinical aspect* of croupous pneumonia differs from that of bovine pleuro-pneumonia by its acute and even peracute course and the typical sequence of its several stages of congestion, red and yellow hepatization and of resolution. And more especially at the beginning of croupy pneumonia, when there is already a high degree of (infectious) fever, no dulness is present, as in bovine pleuro-pneumonia, which, as a rule, runs a chronic and irregular course.

3. The *post-mortem lesions* are essentially different in the two diseases. In croupous pneumonia, owing to the acute course of the malady, the hepatized portions of the lungs are alike in age, colour and condition ; consequently, are perfectly uniform. The interstitial lymphatic system is either not implicated in the process at all, or only oedematously infiltrated, or, in extreme cases, permeated by a thin, fibrinous exudation, with isolated white blood-corpuscles. On the other side, the

characteristics of bovine pleuro-pneumonia are: first, the varying age and appearance of the several foci of hepatization, and, secondly, the preponderant participation of the interlobular lymph-spaces. This last manifests itself in extreme inflammation of the interstitial lymph-vessels (*lymphangioitis*), with formation of thrombi inside the vessels and great inflammatory infiltration of the interstitial connective tissue, by which the latter is broadened. In consequence of this the pleura is almost always sympathetically affected in the form of exudative fibrinous pleuritis.

From the pneumonia which occurs in the course of deer and cattle disease, the above inflammation of the lungs is distinguished by absence of contagion.

**Marbling of the Lungs.**—Great importance has always been attached to so-called marbling hepatization as a distinguishing sign. By Gerlach this was claimed exclusively for bovine pleuro-pneumonia, whereas, according to others, it is said to be also present in non-contagious croupous pneumonia. It here becomes a question of what we understand by marbling hepatization. Those who consider a mixture of yellow or yellowish-grey bands (infiltrated inter-lobular cellular tissue) with red patches (hepatized lung-lobules) as marbled hepatization, will find the same in several diseases of the lungs, particularly in pneumonia medicamentaria. But if, on the contrary, we only regard as marbled hepatization an aspect in which the various lung-lobules present varieties of age and of degrees of hepatization (the central lobules of the affected part usually show the older changes), and in which the same are fringed and interwoven by inflammatorily infiltrated connective tissue, then we must agree with Gerlach and admit that this condition is found only in chronic bovine pleuro-pneumonia, and not in the croupous pneumonia of cattle running with acute course. The variously coloured, brownish, yellowish and grey patches, surrounded by interstitial bands, are therefore alone the decisive indications.

**Therapeutics.**—Medicinal treatment is rarely required, owing to the rapid course of croupous pneumonia. It is, however, naturally quite the same as that indicated for the similar condition in horses. Cold wrappings and infusions, as well as administration of anti-pyretics, especially alcohol, are equally appropriate for cattle.

(c).—CROUPOUS PNEUMONIA IN THE SMALLER DOMESTIC ANIMALS.

1. In **dogs**, according to Röll, croupous pneumonia is frequent and usually attacks the posterior lobes of the lungs. But we cannot confirm this statement, and must rather assert that true croupous, lobar pneumonia is not in general common among

dogs, and, in comparison with the pneumonia of distemper, is somewhat rarely met with. The clinical aspect and pathological condition correspond exactly with those described in the horse. The commonest form of pneumonia in dogs is the catarrhal.

2. In **pigs** the complaint is relatively commoner, especially among such as are driven long distances in great heat or during severe cold. But, as compared with infectious pleuro-pneumonia (swine-plague), the malady seems to be rare. But exact descriptions are here also lacking.

3. Among **sheep** Roloff often met with acute croupous pneumonia in very young lambs, from 14 days to 3 weeks old. The inflammation almost invariably attacked the anterior lobe of one or both lungs, and the animals mostly died at the height of the disease from lung-oedema of the non-hepatized parts of the organ. Cox reports in a similar sense.

4. We have noted the disease somewhat more frequently among **cats** than among dogs.

#### 2.—CATARRHAL PNEUMONIA. BRONCHO-PNEUMONIA.

**Etiology.**—The causes of catarrhal pneumonia are in general those of acute bronchitis. Besides cold, all objects which cause irritation and which reach the lungs with the inhaled air, such as dust, sand and other foreign objects, fungi, and infective matter must be included. Broncho-pneumonia, when due to the latter causes, often appears in an enzoötic form, particularly among young animals (calves, lambs, sucking pigs). From acute bronchitis a broncho-pneumonia is developed either by spread of the inflammation from the bronchioli to the alveoli and inter-lobular tissue, or by aspiration of the infectious bronchial secretion into the alveloi.

The complaint appears most frequently as an accompaniment of infectious diseases, especially such as attack young and feeble animals. In the first place stands the distemper of dogs; the catarrhal pneumonia developed during its course is the most common pneumonia of dogs. It is also fairly common in cattle, and is of great importance because it forms the usual foundation for the entrance and spread of the tubercle bacillus. In horses it is rarer, but not unknown. Among sheep, pigs and calves, as well as poultry, it plays an important part in the form of worm-pneumonia.

A special form of catarrhal pneumonia is the hypostatic or sinking pneumonia. It arises when, in consequence of gradually increasing weakness of heart, the flow of blood from the lungs is disturbed; its opening stage is therefore one of hyperæmia of the lungs. Hypostatic pneumonia has its seat in the lower parts of the lungs, whither the congested blood sinks by gravity. It occurs especially in weak and enfeebled individuals, and is commonest in sheep; also during chronic heart-complaints, blood-diseases, tetanus, etc.

**Pathological Aspect.**—Catarrhal pneumonia is a strictly localised and therefore usually a lobular lung-inflammation. Sometimes there is only one isolated area, but frequently many areas become fused, and under some circumstances the broncho-pneumonia may become disseminated and present a great number of very small miliary foci of inflammation. This form of pneumonia also begins with hyperæmia and haemorrhage, which are complicated with exudation, emigration of white blood-corpuscles and increased desquamation of epithelium. There is no excretion of fibrin. Consequently, on microscopical examination of the alveoli, they are not found filled with a net-like coagulation of fibrin, as is the case in croupous pneumonia. According to the preponderance of the several inflammatory products, the lungs are found on section to be at first black to dark-brown, and later, as the cellular elements increase, a greyish-red, greyish-yellow, and at last yellowish white colour. Corresponding hereto, pressure upon the alveoli and bronchioli produces at first a bloody or dull chocolate-coloured fluid, which is later greyish-red to pale grey. In contrast to the coarsely granulated section-surface in croupous pneumonia, the surface is here smoother, or at most only finely granulated. The consistence of the inflammatory foci is firm and hard, the tissue itself contains no air and projects slightly above the section-surface. The surrounding healthy lung-tissue is in a condition of compensatory hyperæmia. If the broncho-pneumonia be disseminated, we find scattered over the lungs whitish-grey pus-foci as large as a poppy or hemp-seed, or larger. Sometimes diffuse purulent infiltration of larger lung-areas is also observed.

Among the conditions which follow catarrhal pneumonia atelectasis of the lungs is most important. It arises partly from stoppage of the bronchioli and alveoli with exuded matter. In the airless portions of the lungs haemorrhage first sets in

owing to congestion, followed by adhesion and growing together of the alveolar walls, as well as fresh growths of connective tissue with thickening of the septa (collapse-induration). The atelectatic spots are marked by a dark bluish-red colour. A slaty-grey pigment is formed out of the haemorrhages (slaty induration). Sometimes the surface of the lungs sinks in over the site of the atelectasis. The lung-tissue round about the broncho-pneumonic foci, as well as that between them, is often vicariously emphysematous. The bronchi also not unfrequently expand and form bronchiectases with chronic inflammatory processes upon the mucous membrane. The pleura above the broncho-pneumonic foci is sometimes rough, the bronchial glands are swollen, oedematous and even purulently infiltrated. Among other consequences we must also mention suppuration and formation of ichor, as well as caseation of the lung-tissue. If foreign bodies have penetrated into the lungs, they are found covered by a more or less thick envelope of connective tissue (nodular, fibrinous induration).

**Symptoms.**—The phenomena of catarrhal pneumonia are at the outset those of acute bronchitis, from which it develops. Then come a rise of bodily temperature to medium or even high fever, acceleration of respiration and also, as a characteristic sign, especially in dogs, a short, painful cough, which is therefore often suppressed. When there are larger broncho-pneumonic foci superficially placed, percussion reveals also circumscribed, lobular, often quite localised dulness, especially along the lower edge of the lungs, upon whose periphery a tympanitic tone is often heard. Contrary to the conditions in croupous pneumonia, auscultation very seldom discovers bronchial breathing, but rather the breathing sounds at the dulled places are either much weaker or have altogether disappeared. On the other hand, several kinds of rattling noises can be heard.

The course of the condition is at times very rapid, but is in general very slow and chronic, extending over weeks and months. The result is either recovery after two or three weeks in consequence of resorption and expectoration of the inflammatory products, by which the fever abates, breathing and cough become easier and the rattling sounds grow moister; or else the disease changes to chronic conditions, among which chronic induration of the lungs (lung-asthma), with its later complication, phthisis of the lungs, is the principal one. Sup-

puration and necrosis of the lungs develop more rarely, and are known by high fever, great emaciation and weakness, increasing stupor and cessation of the cough. But death may occur acutely as a result of the inflammatory process spreading to larger divisions of the lungs, or of secondary œdema of the lungs. The prognosis is particularly bad with young and feeble individuals.

**Differential Diagnosis.**—Catarrhal pneumonia is distinguished from the croupous form by its slower and atypical course, its development from a bronchitis, its lobular dulnesses, by the absence of a rust-coloured nasal discharge and by its greater malignity. Moreover, it is in horses the rarer form, but in dogs the common type. It may be more difficult to know it from capillary bronchitis (bronchiolitis); indeed, one cannot always fix exactly the time when the latter changes into broncho-pneumonia. In general the decisive indications are, higher fever, a painful cough, localised dulnesses and the absence of vesicular breathing sounds. But only too frequently these most valuable signs of catarrhal pneumonia are not always fully present. For instance, it may be impossible to prove a dulness, because the broncho-pneumonic foci lie too low down or are not large enough, so that an opinion is difficult to form.

**Therapeutics.**—The treatment of catarrhal inflammation of the lungs agrees in the main with that of acute and chronic bronchitis. But anti-pyretic remedies are more frequently used, as well as those which have a tonic action upon the heart, and for the latter purpose we recommend, besides alcohol, especially digitalis and caffein. When the course is chronic, use loosening and expectorant remedies (alkalis, neutral salts, apomorphine, ethereal oils, resins). Inhalations are also much used, especially of creolin, and likewise hydropathic fomentations. Attention must also be paid to diet.

Friedberger has described an enzoötic catarrhal pneumonia in *lamb*, which had a certain likeness to swine-plague. Its signs were a feverish pneumonia, with widespread dulness of the lungs upon one side. *Post-mortem* revealed lobular catarrhal-desquamative foci of inflammation, along with extensive catarrhal pneumonia and multiple caseous necrosing foci. Furthermore, pleuritis, purulent-fibrinous pericarditis and localised necrotic hepatitis and peri-hepatitis were all in evidence. The causes of the disease remained unknown.

Galtier asserts that he has seen swine-plague communicated to wethers.

**3.—PNEUMONIA DUE TO FOREIGN BODIES. TRAUMATIC PNEUMONIA. GANGRENE OF THE LUNGS.**

**Etiology.**—The pneumonia produced by foreign bodies is at first nothing else than catarrhal, lobular, localised pneumonia. But as it has ever been regarded as a separate form of the disease, owing to the peculiarity of its causes, we have here adopted the same rule purely on practical grounds. We distinguish two kinds of this pneumonia; viz., that which is due to foreign bodies in the narrower sense, and traumatic pneumonia.

1. **Pneumonia due to foreign bodies** in the narrower sense is due to such outside matter as enters the lungs by way of the air-tubes and bronchi. In its early development it presents a lobular catarrhal pneumonia, but soon advances to extreme conditions of inflammation, viz., to croupous exudation and finally to gangrene of the lungs. We class here the pneumonia caused by inhaling dust, hot smoke, pungent gases, by the penetration of corn-ears, twigs, pig-bristles and other foreign objects, as well as by worms and mould-fungi. To this form of pneumonia belongs also that very important class of cases known as swallowing pneumonia and pneumonia medicamentaria (drenching pneumonia).

The pneumonia caused by wrongly swallowing is commonest in horses, but also occurs in cattle. Among horses, pharyngitis, diseases of the gullet, cerebral affections and tetanus all provide occasion for the penetration of fodder into the larynx and trachea. Among cattle its most frequent cause is the paralytic form of puerperal fever (puerperal paresis). But also in foot-and-mouth disease, whenever an aphthous pharyngitis develops, a swallowing pneumonia may be set up, as well as in coughing during rumination, or after large doses of physostigmine and pilocarpine. In all animals pneumonia due to foreign bodies may arise by the breathing of the foetus while still in the uterus, if the amniotic fluid or meconium happen to penetrate to its lungs. The lung-inflammations of new-born animals, when not of pyæmic origin, may probably be mostly traced to this cause. Moreover, the pneumonias arising from spontaneous breaking-up of retro-pharyngeal abscesses, or from the gradual settlement of putrid bronchial secretion in the lower-lying portions of the lungs, belong to this category, as does likewise

the artificially produced vagus-pneumonia (paralysis of the laryngeal nerves, entrance of food into the larynx and windpipe).

But more important than these is the drenching-pneumonia or pneumonia medicamentaria, especially in pigs, horses and cattle. Its cause is to be sought in the faulty administration of drugs, particularly liquids. To administer medicine through the nose, as is customary with quacks, to pull out the tongue, to close the nose, to strap up the throat tightly, to pour in too large a quantity at once, continuously to hold up the animal's head, are all actions likely to result in pneumonia. The same is the case with the use of mixtures which require stirring (vegetable powders, infusions of flowers, irritant, nauseous or viscid and stringy fluids). In pigs also one has the animal's often quite incurable obstinacy and screaming to contend against. With horses it is particularly dangerous to continue to hold up their heads to administer medicine after they begin to cough. Finally, intra-tracheal injection, which has lately become customary, may be followed by gangrenous broncho-pneumonia, especially if too concentrated a solution be used.

The further reaction of the bronchial mucous membrane and of the lung parenchyma depends upon the nature of the offending foreign body. Whereas distilled or spring water, as well as all non-irritant drugs in the form of solutions, can be re-absorbed without causing any subsequent inflammation, the intrusion of insoluble, of irritant or even corrosive substances produces various degrees of irritation, from the simple catarrhal to the diphtheritic necrotic and gangrenous.

2. **Traumatic pneumonia** belongs partly to the province of surgery. In this form of the disease the foreign body penetrates into the lungs from the outer surface of that organ. In cattle this occurs oftenest during traumatic pericarditis, and in dogs through needles which work their way from the gullet to the lungs. But in this latter affection pleuritis is more prominent. Here also we must mention the so-called contusion pneumonia arising after crushing the thorax by a blow or a fall, as well as lung-injuries inflicted by rib-fractures, external wounds, etc.

**Gangrene of the Lungs.**—Pneumonia due to foreign objects is one of the most frequent causes of gangrene of the lungs. But the latter may develop in other ways, and especially in horses during murrain

(necrotising pneumonia), after severe, croupous pneumonia with impeded circulation, after hemorrhagic stoppages, in connection with embolic processes (compare the paragraphs on metastatic pneumonia), with bronchi-extasis and stagnant decomposing bronchial secretion.

**Post-mortem Lesions.** — Pneumonia due to foreign bodies presents pathologically a mixture of catarrhal, croupous, necrotic and gangrenous pneumonia (gangrene of the lungs). This last is a combination of necrosis and gangrene and therefore really a putrescent or bacterial pneumonia. At first the inflammatory process is restricted to the bronchi, but gradually seizes the peri-bronchial connective tissue and the parenchyma of the lungs, which latter shows successively the phenomena of catarrhal, croupous (especially in cattle) and diphtheritic inflammation. The affected foci in the lungs are at first blackish-red, then greyish-red, and later a muddy yellow, possess increased resistance, are almost void of air and project very slightly above the section-surface. It is very rarely possible to detect the foreign body itself in the middle of the lobe. Gradually then a suppurative infiltration is developed in the portion attacked, which breaks down the tissue and often appears in the form of multiple abscesses, which lie together in groups, readily coalesce and become foci of gangrene when air enters from the bronchi. The drenching-pneumonias are usually bi-lateral and settle mostly in the neighbourhood of the base of the lungs, i.e., of the principal bronchi.

As a result of gangrene the lung-tissue changes into a blackish or grey, tinder-like, greasy substance, at first solid, but later of liquid character, so that at last the lung parenchyma is replaced by a discoloured, stinking ichor, which contains the several products of decomposition of the albumen and fat (leucine, tyrosine, fatty acids, ammonia, sulphuretted hydrogen) and also of organic constituents, enormous numbers of putrefactive bacteria, shreds of parenchyma, pus-corpuscles, detritus, crystals of triple phosphate, etc.

The lung-tissue around these gangrenous foci is in a state of hyperæmia and oedema. In horses we also find occasionally a characteristic swelling of the inter-lobular connective tissue, in the form of broad, yellow, gelatinous bands, analogous to bovine pleuro-pneumonia. The pleura often shows a purulent or haemorrhagic-ichorous inflammation; moreover, pneumo-thorax and pyopneumo-thorax are not rare. If the gangrene last long, a demarcatory lung-inflammation is formed around the gangrenous focus. The mucous membrane of trachea and

bronchi is sometimes purulently or ichorously inflamed, the healthy lung is often emphysematous, and we have even observed sub-pleural, mediastinal and also subcutaneous emphysema. The rest of the body shows the aspect of sepsis or pyæmia, viz., tar-like blood, swelling of the most important organs, haemorrhage and metastases.

**Symptoms.**—The phenomena of pneumonia due to foreign bodies are at first those of a localised catarrhal pneumonia and may easily be overlooked. Not until gangrene of the lungs begins to develop (lung-caverns) do the more pregnant signs appear. These are :

1. The *breath* has at first a sweetish odour, then *smells badly*, and finally has an intense stench, which is noticeable from both nostrils and is often conjoined with a discoloured, stinking nasal discharge, in which are shreds of tissue. Nevertheless, this bad smell may in solitary cases be absent, although the gangrene be far advanced.

2. *Physical evidence of the presence of cavities.* When these exist and are not too deeply seated, percussion reveals a tympanitic note like the noise of a cracked pot or else a metallic ring. On auscultation we perceive rattling sounds, bronchial breathing, amphoric respiration and splashing.

3. *High fever, with shivering and very frequent, small pulse* (in horses from 80 to 120 a minute), combined with weakness, debility, sopor and profuse diarrhoea, all signs which are characteristic of septic fever. But we must here remark that, according to our experience, although only in solitary cases, appetite may continue good and there may be hardly any fever until just before death, although extensive caverns exist in the lungs.

**Diagnosis.**—From the foregoing the following data are necessary for the identification of lung-gangrene, viz., the proof of a gangrenous odour and of the existence of caverns. To these comes a microscopical examination of the nasal discharge for elastic lung-tissue, or for any of its parenchyma ; the presence of the latter assures diagnosis in all cases. As already stated, the disease is difficult to recognise at the outset, unless its causes be known. On this point one must remember that all acute lung disorders, which develop as complications of colic, pharyngitis, inflammation of the brain, puerperal paresis, tetanus, petechial fever, diseases of the gullet, etc., are open to grave suspicion of being pneumonia due to foreign objects.

**Differential Diagnosis.**—It is possible to confuse pneumonia due to foreign bodies and lung-gangrene with bovine pleuro-pneumonia, bronchiectasis, phthisis, diseases of the teeth and cranial bones and affections of the sinuses. But the two last-named maladies may be known by the fact that the breath from one nostril alone smells badly. Moreover, when the teeth are diseased, it is the oral fluid which smells especially. More difficult and often impossible is the distinction of a lung-cavern from a bronchiectasis; but in the latter there are no elastic fibres in the nasal discharge, nor is there usually the high degree of sepsis. From bovine pleuro-pneumonia that due to foreign bodies can be known by its acuter course, its tendency to gangrene and the absence of true marbling.

**Prognosis.**—This varies with the quantity and also with the chemical and physical nature of the intruded foreign body. In general, it is unfavourable, for by far the majority of affected animals perish. Death then occurs in a few days, or sometimes not for several weeks. Cure is nevertheless not rare, even when the gangrenous smell has made its appearance. It is quite possible for a gangrenous focus to be cast off by suppuration and expectoration. Such recoveries seem to occur especially in cattle. But if once the gangrene become diffuse, instead of merely local, then recovery is hopeless.

**Therapeutics.**—The treatment of pneumonia caused by foreign bodies is the same as for the catarrhal form, but when gangrene has set in becomes almost fruitless. In this case one can merely try to check the gangrenous process by inhalations or by intra-tracheal injection of creolin, lysol, ichthyol, tar, carbolic acid, oil of turpentine, etc., remedies which may also be administered internally. At the same time the septic fever must be combatted with anti-pyretics, especially camphor and alcohol. As a preventive, great care in the administration of fluid medicines is imperative, as well as the avoidance of all drenching for pigs.

#### 4. MYCOTIC INFLAMMATION OF THE LUNGS IN MAMMALIA.

##### *Pneumo-mycosis aspergillina.*

**Etiology.**—Mycotic inflammation of the lungs may be produced by various fungi: mould-fungi, actinomycetes, botryomycetes and schizomycetes. That caused by mould-fungus is

described as mycotic inflammation of the lungs in the narrower sense. Among mammalia (horses and cattle) it is mostly *aspergillus nigatus* which enters their bodies with mouldy food, or by inhalation of the spores; but among birds (fowls, pigeons, geese, ducks, parrots, cage-birds, swans, pheasants and francolines) it is mainly *aspergillus glaucus*, *nigrescens* and *tumigatus*, likewise *mucor racemosus* and, more rarely, *conoideus*. Feeble or tender individuals, or such as suffer from catarrh, seem to be most liable to infection by mould-fungus. The disease often occurs quite epidemically, especially among poultry. But Pech once saw seven horses in one stable affected with this disease after eating mouldy, musty straw-chop. Fuller details respecting the mycotic pneumonia of poultry are given in Chapter XXVI.

Although it must be admitted that in a great many cases where mould-fungi are found in the lungs, such as in bronchiectasis, caverns and pneumonia due to foreign bodies, their presence is merely accidental and causes no pathological injury, yet it has been clearly shown in many carefully observed instances that they have an injurious effect. By mechanical and chemical irritation, as well as by consumption of the neighbouring lung-tissue, these mould-fungi cause intensely inflammatory processes in the alveoli and bronchioli and also on the mucous membrane of the bronchi, which much resemble those described in the pneumonia due to foreign bodies.

**Pathological Aspect.**—In mammalia pneumo-mycosis usually presents the appearance of a nodular, suppurative pneumonia. The several nodules are from a hemp-seed to a lentil or pea in size, and are partly spread in great numbers over the lungs and partly confluent. They are made up either of a connective-tissue capsule, with a purulent centre containing fungi, or of globular and very small foci of inflammation, the centre of which is occupied by a broad field of mould, and their periphery separated from the sound tissue by a haemorrhagic or hepatized zone. We only rarely find diffuse inflammation of the lungs, but owing to hepatization and inflammatory infiltration of the inter-lobular connective tissue, this may in cattle have a deceptive resemblance to bovine pleuro-pneumonia. Microscopical examination will supply the decisive data. Likewise in horses this nodular pneumo-mycosis may sometimes lead one to suspect glanders. Upon the bronchial mucous membrane we may also observe ulcers with fungoid proliferation.

The pleura above the affected parts of the lung is frequently in a state of pleuritis sicca. In one case Martin demonstrated, along with the lung abscesses, also abscesses in the liver, which contained fungi. In very acute course the complete aspect of pneumonia due to foreign objects may develop, with consecutive gangrene of the lungs.

**Symptoms.**—The phenomena correspond with those of catarrhal pneumonia. Extensive lobular dulness on percussion seems to occur more rarely. Thus in a very acute case in a horse, which was complicated with gangrene of the lungs, Pech found the dulness to extend over half the thoracic wall. The disease is usually chronic, leading to an appearance of phthisis of the lungs, with growing difficulty of breathing and emaciation. Occasionally masses<sup>1</sup> of fungi can be discovered in the sputum, a fact naturally important for diagnosis.

**Therapeutics.**—It is generally difficult to commence any treatment for pneumo-mycosis, seeing that its causes can rarely be known until after death. Moreover, the fungi, when once entered, cannot well be removed or destroyed. The utmost that can be done is to attempt inhalations of vapours of tar, carbolic acid or turpentine, but they are not likely to do much good. For larger animals intra-tracheal injections of creolin, carbolic or salicylic acid or sublimate (mixed with water) may be experimentally tried, or a sufficiently dilute Lugol's solution, as in worm or verminous pneumonia. The disease has, however, more pathological than clinical interest.

##### 5. INTERSTITIAL PNEUMONIA. PHTHISIS OF THE LUNGS.

**Etiology.**—This is not an independent disease, like croupous or catarrhal pneumonia, but the new formation of connective tissue which characterises it is connected secondarily with chronic inflammatory processes in the lungs and bronchi. Among these comes first catarrhal pneumonia, which during its later course almost always shows new growth of connective tissue; then come the pneumonia caused by foreign bodies, and also chronic bronchial catarrh, with its very frequent bronchiectasis. But above all there are certain infectious pneumonic affections which are accompanied by great increase of connective tissue, such as tuberculous, glandrous, actinomycotic and bovymycotic pneumonia, bovine pleuro-pneumonia and the

distemper of dogs. Croupous pneumonia of horses also, though but rarely, leads sometimes to new formation of connective tissue. In spite of its merely secondary importance, we here treat interstitial pneumonia in a separate section for the sake of greater clearness.

**Pathological Aspect.**—Interstitial pneumonia consists in the formation of new connective tissue in the lungs, in the form of nodules between the several alveoli and lobules, or round about the bronchi. The parts of the lungs thus degenerated into connective tissue are firm in texture and creak when cut with a knife; their volume is usually diminished and they are white to greyish-white in colour. The connective tissue follows a gelatinous and soft infiltration, which compresses the alveoli and bronchioli, so that their walls even grow together; it then gradually shrinks and hardens (connective-tissue induration, or sclerosis of the lungs). In consequence of this the conducting bronchi often expand (bronchiectasis), as do also, sympathetically, the unaffected alveoli (emphysema).

**Symptoms.**—The chief of these is dyspnoea (in horses asthma), provided that a considerable part of the lungs be lost for breathing purposes. Owing to the frequent presence of chronic bronchial catarrh, there is also often a short, superficial and faint cough. It is not seldom the case that no physical change can be demonstrated by percussion or auscultation, because the foci are very small or scattered, or else lie in the deeper layers of the lungs. In other cases we obtain dulness and the further symptoms of catarrhal pneumonia. If of longer duration, interstitial inflammation of the lungs produces gradual emaciation and general chronic illness, combined with debility, dropsy, albuminous urine, etc., symptoms which, along with the accompanying dyspnoea, are known as lung-phthisis (phthisis pulmonum). Death occurs in such cases from exhaustion.

*Treatment* is usually unavailing. At best one may try alkalis or iodide of potassium.

**Lung-phthisis.**—This name is applied to all chronic, destructive changes in the lungs, accompanied by great loss of flesh and of strength. It is therefore a mistake to believe that only tuberculosis of the lungs can lead to lung-phthisis. In cattle (and in man) this is indeed most frequently the case. But other causes may produce it just as easily, such as glandular processes in the lungs, catarrhal pneumonia, pneumonia due to foreign bodies, antinomycosis, tumours in the lungs, etc.

**Lung-tumours.**—As such we must cite carcinoma, melanoma, sarcoma, cavernous tumours, chondro-adenoma and fibroma. They generally avoid clinical diagnosis, as they are very hard to distinguish from other thickenings of the lungs. The metastatic lung-tumours can only be diagnosed with a certain amount of probability, when certain symptoms arise in the lungs in connection with external primary tumours (carcinomata), which lead one to suspect displacement of the cells of the tumour inwards (circumscribed dulness, dyspnoea, lung-phthisis, etc.). In a case of primary lung-carcinoma in a dog we frequently observed haemorrhage in the lungs during life. Grammlich has noticed the same thing in horses.

#### 6. METASTATIC OR EMBOLIC PNEUMONIA.

**Etiology.**—This also is not an independent, but a secondary condition. It occurs when waste matter (emboli) is carried into the lungs from a peripheral vein containing thrombi, or from one discharging into an ichorous focus or from the heart. On becoming wedged into the capillaries such matter causes first a haemorrhagic infarct, setting up, owing to retrograde movement, a congestive hyperæmia in the portion of lung thus cut off from the blood-stream, with accompanying haemorrhage. Should the emboli be septically infected, this becomes a lung abscess with gangrene of the organ. The commonest causes of this condition in our domestic animals are: fistulae resulting from phlebotomy, thrombo-phlebitis on the hinder extremities and between the thighs of horses, gangrenous inflammation of the feet, e.g., gangrene of the fibro fatty frog, suppurative mastitis, abscesses formed during strangles of horses, pyæmic processes starting from various organs, ulcerous endocarditis, etc. After extensive suppurative infiltration of the upper thigh Semmer observed fatty embolism of the lungs in a horse.

**Post-mortem Lesions.**—Metastatic pneumonia is localised. The foci vary from a pea to a walnut in size, and are chiefly found on the upper surface of the lungs corresponding to the distribution of the vessels. The haemorrhagic infarcts represent wedge-shaped (the base of the wedge turned outwards), dark-red, hard and airless foci which, if there be septic matter in their vicinity, undergo suppuration, thus finally changing the entire haemorrhagic plug into a rounded, purulent or ichorous abscess (lung-cavern). Along with the above we find the lesions of pyæmia and septicæmia.

**Symptoms.**—These consist in the sudden appearance of

high fever, shiverings, hard breathing and cough. Sometimes also, though not always, we can detect dulness or a hollow in the lungs (signs of caverns). It is always important to demonstrate the primary focus. The process generally ends fatally from general pyæmia. Only very rarely is a cure effected after ejection of the gangrenous focus. If the ichorous focus break through into the pleural cavity, a suppurative pleuritis (empyema) is then developed.

The *treatment* of metastatic pneumonia is almost useless; one may try the same methods as are suggested for gangrene of the lungs.

HYPERTÆMIA AND OEDEMA OF THE LUNGS.

**Etiology.**—1. **Active Hypertæmia of the Lungs** occurs especially in horses, and in those which are full-blooded and well-nourished, after over-exertion, running too fast, or during excessive summer heat. In other cases the cause is the inhalation of cold or over-heated air, or of pungent and irritant gases. The exclusion of larger sections of the lungs from free circulation of blood, owing to compression by exudates, may also lead to collateral active hypertæmia of the lungs. Finally, it forms the opening stage of pneumonia.

2. **Passive Hypertæmia of the Lungs** is mainly a sequel of such heart defects as produce congestion of blood in the lungs. Among these we reckon all diseases accompanied by great weakness of heart and in which a sinking or hypostatic pneumonia is developed. A similar congestion of blood in the lungs occurs when the abdomen is greatly distended by accumulation of gas in the stomach or bowels, whereby compression is exerted upon the larger vessels.

3. **Oedema of the Lungs** consists in extravasation of serum from the blood-vessels into the alveoli, bronchioli and bronchi, and evidently arises in various ways. Inflammatory oedema of the lungs represents a serous pneumonia, which develops in complication with intense active hypertæmia of the lungs during croupous pneumonia, as well as during certain infectious diseases. Many cases of acute lung-oedema in cattle and sheep seem especially to point to malignant oedema. Thus Kitt observed acute lung-oedema in a sheep in which the *post-mortem* revealed no cause for the general symptoms, but where numerous

bacilli of malignant œdema were found in the juices which escaped from the lungs. The condition of lung-œdema is caused by engorgement of blood in the lungs as well as by inflammation, especially during the death-agony, when the venous blood can no longer flow from the lungs owing to relaxation of the heart. Finally, we must assume abnormal changes and great permeability of the vascular walls as being in many cases the cause of œdema of the lungs.

**Pathological Aspect.**—In *active hyperæmia* of the lungs these organs are found very full of blood, dark-red in colour, of increased volume, consequently bloated and firmer to the touch. Blood flows copiously from the section-surface. The capillaries are fully charged and distended, so that they project far into the lumen of the alveoli. The bronchi contain much frothy mucus, which is more or less stained with blood.

In *passive hyperæmia* of the lungs, if the congestion be of long standing, we have the aspect of a so-called "engorged lung," or of brown induration. The lung is tougher, penetrated by haemorrhages, from which later grey or black pigment-spots develop; the vessels are greatly dilated; the mucous secretions of the bronchi contain many red blood-corpuscles; and, finally, the lungs are partially splenetic.

*Œdema of the Lungs* is characterised by great enlargement and an enormous abundance of juice upon the section-surface, over which flows a plentiful froth of very fine bubbles, which is also found in the bronchi. In addition we find great desquamation of epithelium and extravasation of red blood-corpuscles.

**Symptoms.**—In the first and third of the above three forms, which occur most frequently as independent diseases, the signs usually appear suddenly and unexpectedly. The animals betray great difficulty of breathing. Respiration is quickened and impeded, the number rising to sixty, eighty and even one hundred per minute. The nostrils are widely dilated, the visible mucous membranes very red, even bleeding at the nose and frothy nasal discharge are perceived, whereby the animals grow very restless and timid and are in danger of choking. There is also a superficial, short and, at first, dry cough. In active hyperæmia percussion reveals nothing abnormal, but in œdema we have sometimes a tympanitic tone, or at least a tympanitic accessory note to the loud sound, while in congestive

cœdema (hypostatic pneumonia) we have dulness. In active hyperæmia auscultation detects a much intensified vesicular breathing, and in lung-œdema the sound of crepitation, while, as death approaches, we hear bubbling and rattling noises. The pulse is very quick, full and hard, the heart-beat palpitating. If the condition terminates in death, all the breathing trouble gradually abates and is followed by a state of stupefaction. The course of the malady is always very rapid, not lasting usually above twelve to twenty-four hours. Either there is prompt recovery, speedy suffocation, or a subsequent inflammation of the lungs. Prognosis is nevertheless not unfavourable.

**Therapeutics.**—Liberal blood-letting has a truly life-saving effect in active hyperæmia and in cœdema of the lungs. This is one of the few diseases which call for its prompt performance. One must also attempt cutaneous derivation by friction and blisters, also give laxatives, purging clysters and cold infusions of the rectum.

#### PULMONARY HÆMORRHAGE. SPITTING OF BLOOD. HÆMOPTYSIS.

**General Note.**—Discharge of blood from the lungs is not a disease of itself, but an accompaniment and sequel of various diseases. Owing to the striking nature of its phenomena it has attracted much attention, which may perhaps justify us in devoting a special section to its consideration. The condition is not generally very common, but occurs oftenest in horses and cattle.

**Etiology.**—In most cases of hæmoptysis the blood comes from the tissue of the lung itself, more rarely from the mucous membrane of the bronchi, and still more rarely from the larynx. The causes are various. In horses over-exertion plays the chief part. Dragging excessive loads, riding too far or too fast, shock and exhaustion after bolting are all occasionally followed by a discharge of blood. Then, too, hæmorrhage of the lungs is somewhat frequently produced in horses by glanders, and in cattle is so far caused by tuberculosis as that, during the suppuration and formation of cavities in the lung-tissue, larger vessels are thereby corroded and opened. Hæmorrhage may also occur in the parenchyma of the lungs in the course of hyperæmia of that organ, particularly the passive form, and also during the contagious pleuro-pneumonia of horses. Emboli

also lead not rarely to haemorrhagic congestion and emission of blood from the bronchioli. Finally, haemoptysis is caused by certain arterial diseases, rupture of aneurysms, worm-knots (*strongylus armatus* in horses), or ulcerative new growths in the lungs. This symptom is also observed during many infectious diseases, such as anthrax and septicæmia. Haemorrhage caused by external injury cannot suitably be discussed here.

When it occurs in the bronchi and larynx, its cause is traceable to foreign bodies, or to tuberculous or glanderous ulcers.

**Symptoms.**—The characteristic sign of haemoptysis is the discharge through the nostrils and mouth, and often in large quantities, of bright-red, frothy blood. This frequently gushes out in a stream. At the same time considerable difficulty of breathing is observed. The animal is in danger of suffocation, breathes violently and rapidly, coughs much, trembles, appears restless and troubled, sweats profusely and begins to sway and stumble when the loss is serious. In such cases the mucous membranes grow continually paler, the skin turns cold, the pulse is small and at last imperceptible. On auscultation we hear bubbling sounds in the trachea and bronchi. Sometimes there is only one such attack, but very frequently the bleeding is renewed after several hours or days. Death may occur suddenly from loss of blood, but the animal not seldom recovers, and the bleeding ceases permanently. Respecting differentiation from the vomiting of blood compare the differential diagnosis of Hæmatemesis, Vol. II., page 160.

**Therapeutics.**—Treatment is regulated entirely by the exciting cause. Blood-letting can only be adopted when this is either hyperæmia or inflammation of the lungs. In all other cases it does harm. Cold fomentation of the thorax may always be applied, and one may try the internal administration of styptic remedies : ergot, hydrastis, tannin, alum, sugar of lead, sulphate of iron, etc. Inhaling the vapour of chloride of iron and of vinegar is said sometimes to bring relief. As a prophylactic avoid every excitement or movement, too much warmth and all irritating fodder.

#### EMPHYSEMA OF THE LUNGS.

**Etiology and Pathogenesis.**—We have here the presence of an abnormal amount of air in the lungs, which may arise

in different ways. Its causes are most frequently purely mechanical, the entire lung, especially in working horses, being abnormally enlarged owing to excessive distension of the thorax after forced inspirations ; or it may be that only certain divisions thereof are affected, because others, being inflamed or in a state of atelectasis, are impermeable. Changes in the texture of the lung-tissue after inflammation, such as pneumonia or bronchial catarrh (inflammatory emphysema), or due to old age (senile emphysema), are also frequently causes. In many cases there appears to be a certain predisposition of the lung-tissue to destructive, parenchymatous conditions of involution, such as attenuation of the partition-walls (atrophic emphysema). That which arises from external injury (traumatic emphysema) belongs rather to the department of surgery. Finally, putrid gases (hydrogen, carburetted hydrogen, sulphuretted hydrogen) may be generated in gangrenous lung-foci, such as arise during pneumonia caused by foreign objects, infectious pleuro-pneumonia, etc., and these may emphysematously distend the lungs (septic emphysema).

The following forms of emphysema may be clinically distinguished :

1. Chronic or substantial.
2. Interstitial or inter-vesicular.
3. Acute vesicular emphysema.

**I. Chronic or substantial** emphysema arises through enlargement of the alveoli and obliteration of the alveolar septa, owing to continuous increased air-pressure combined with a degenerative change in the parenchyma of the lungs. The alveoli are first unduly distended, and then larger air-cavities are formed by the union of several such alveoli. The more the alveoli expand, the thinner their septa naturally become. According to Stömmers measurements (to whom we owe the first exact description of a horse's lungs when in this condition), the size of these alveoli is increased tenfold, or from  $\frac{1}{10}$  to  $\frac{1}{8}$  inch, while their walls are correspondingly attenuated (from 8 micro-millimètres to 1 or 2 micro-mm.). The rarefied septa then represent merely small ridges projecting into the interior of the cavity. Great rarefaction is also found in the vascular system of the lungs. In consequence of increased intra-alveolar pressure the alveolar septa are exposed to compression from both sides, whereby the vessels are stretched and narrowed, while the vascular network is widened. For this reason the

capillaries show a strikingly extended course during emphysema of the lungs, considerable contraction of their lumen and often distinct atrophy. We then perceive either broken, rounded stumps, or else two pointed vessel-stumps joined together by a thin cord (vascular atrophy within the septa). This vascular atrophy is the immediate preliminary stage of septum-atrophy. The elastic fibres simultaneously disappear and the epithelium suffers fatty degeneration. Viewed in the mass, the lung, or its affected part, is enlarged, very bulky, poor in blood and juices, pale, very soft, puffy and downy to the touch; its edges are blunt, the surface shows impressions of the ribs and retains finger-prints; when the thorax is opened the lungs do not collapse, nor does its tissue sink when cut. This decay of the lung-capillaries is at the same time associated with a compensatory hypertrophy of the heart.

**The Relation of Emphysema of the Lungs to Asthma in Horses** has been satisfactorily cleared up by the investigations of Stömmer. In nine asthmatical horses he invariably discovered the presence of chronic lung-emphysema and thereby confirmed the opinion previously advanced by Gerlach, Haubner, Bruckmüller and others, according to which asthma is primarily caused by emphysema of the lungs. He has shown that the lungs of horses suffering from asthma are anatomically and histologically like those of men when attacked by chronic vesicular emphysema, and that a horse's lungs are more subject to this condition than those of a man. Moreover, in horses emphysema of the lungs represents to some extent a disease resulting from their avocation (draught-horses, race-horses).

**2. Inter-vesicular or Interstitial Emphysema**, a form somewhat common in our domestic animals, arises from rupture of the alveolar walls by abnormal air-pressure and irruption of air into the inter-alveolar connective tissue and lymph-cavities. We find it most frequently upon the surface of the lungs immediately beneath the pleura (sub-pleural emphysema), which is often uplifted in blisters the size of a pea or walnut (bulbous emphysema). These blisters can be easily pushed away, differing in this from substantial emphysema. The air frequently penetrates from the sub-pleural space towards the root of the lungs and between the laminae mediastinae, attaining finally the anterior aperture of the chest outwards as far as the looser connective tissue about the neck, whence it can spread in the subcutaneous cell-tissue over the whole body, not even omitting the ears and tail. This inter-vesicular lung-emphysema with subsequent subcutaneous emphysema occurs very often in

domestic animals, especially cattle and horses. Its causes are usually over-exertion, drawing excessive weights, especially in great heat or great cold or in face of a high wind, exhausting races, bolting, long and wearying transport, violent coughing-fits and, in cattle, exceedingly frequent, irritant infusions and the cough thereby produced. It may also arise from medicines being swallowed the wrong way and getting into the lungs (mixtures requiring to be shaken), which may become dangerous from the perforative action of acute and acrid vegetable particles. Finally, a localised inter-vesicular emphysema occurs sometimes during the death-agony, owing to increased air-pressure in the lung.

3. **Acute Vesicular Emphysema** is nothing else than simple ectasis of the alveoli, or distension of the same beyond the normal, without anatomical change of structure. Sometimes it spreads over the entire lung, and at others only affects certain lobes. Total vesicular emphysema occurs during the death-agony, and also as an introductory stage of the inter-lobular form. Partial emphysema often occurs when other parts of the lungs become impermeable, especially during chronic bronchial catarrh. The several lobules are inflated, project above the rest of the lung and are very pale. This form of emphysema has no clinical importance.

**Symptoms.**—1. The phenomena of *chronic emphysema* of the lungs are those of asthma, viz., those of chronic, and chiefly expiratory dyspnoea, which is produced on the one hand by the diminished elasticity of the lungs and accumulation of air in the distended alveoli, and on the other by atrophy of the capillary system in the lungs. During its course hypertrophy of the heart is secondarily produced. Compare the several clinical symptoms of emphysema in the next section on "Asthma."

2. The symptoms of *acute inter-lobular emphysema* of the lungs are very characteristic. The animals are suddenly stricken with severe difficulty of breathing and seem to be in danger of choking. Their respiration is anxious, groaning and laboured; cattle open their mouths wide, stretch out the tongue and make painful movements of the muscles of the belly (expiratory dyspnoea). There is no cough, or it is short, superficial and painful. Physical examination of the lungs reveals a very loud, clear percussion ring with tympanitic after-note; auscul-

tation shows peculiar crackling, rustling crepitations and bubbling murmurs, also dry, rattling sounds. We also very often notice the appearance of a subsequent emphysema of the skin, especially in cattle. Appetite and thirst diminish, as also does peristalsis, but there is no feverish rise of temperature. The course of the malady is sometimes very acute, and death from suffocation may occur in from twenty-four to thirty-six hours. In other cases it is rather chronic, and recovery takes place after weeks or months.

*Treatment* can only be attempted when there is also a subcutaneous emphysema. In such cases we recommend massage of the emphysematous parts of the skin, as well as compression with bandages.

**Pneumatoisis of Cattle.**—Michels describes by this name an emphysema of the lungs appearing enzootically among cattle in the swampy districts of the Netherlands, of the origin of which nothing certain is known. Demeester, who had previously observed it in Flanders, traces it, not without reason, to a bronchial catarrh with cough caused by cold when grazing. *Post-mortem* reveals a vesicular, inter-lobular and sub-pleural emphysema.

#### BROKEN WIND. ASTHMA IN HORSES.

**Definition.**—Asthma is rather a legal than a clinical conception. It is generally defined as a chronic, non-febrile and usually incurable difficulty of breathing, which is not directly fatal, and is in no way connected with the nature of the work done. It is met with oftenest in horses, especially those above six years old, but is by no means rare in cattle, dogs and other domestic animals. Other synonyms for the disease are "hard-breathing," "broken-wind," "pursiness," "shortness of breath," etc. We shall here discuss it so far as the condition has any clinical interest. For other details we refer our readers to Gerlach's "Handbook of Forensic Veterinary Medicine."

**Etiology.**—This is far from being uniform. The name is rather a collective title for pathological conditions of very varied origin. The three following merit our chief consideration : 1. Diseases of the lungs ; 2. Diseases of the respiratory passages ; and 3. Diseases of the heart.

**I. Diseases of the Lungs.**—The most important of these is chronic, substantial emphysema of the lungs. But asthma

may also develop in the course of chronic bronchial catarrh with its resultant sequels : bronchiectasis, atelectasis, connective-tissue induration of the lungs, partial formation of emphysema and peri-bronchitis. Of especial importance is capillary bronchitis (bronchiolitis) with its results. The causes of the dyspnoea are here to be sought in narrowing of the air-passages and diminution of the respiratory surface on the one hand (inspiratory dyspnoea), and on the other hand in a decrease of the elasticity of the lung owing to morbid changes therein (expiratory dyspnoea) ; in many cases the latter plays the chief part. Pursiness is also caused by tumours in the lungs : sarcoma, carcinoma, glanderous new growth, etc. ; also by adhesion of the lungs to the ribs, or by connective-tissue induration and atrophy of the lungs following on pneumonia. Finally, compression of the lungs by hydro-thorax, or by hernia of the diaphragm, and abnormal enlargement of the liver or spleen have universally been regarded as causes of asthma, which is also very probable, as the hard breathing thus developed is both chronic, feverless, and on the whole also incurable.

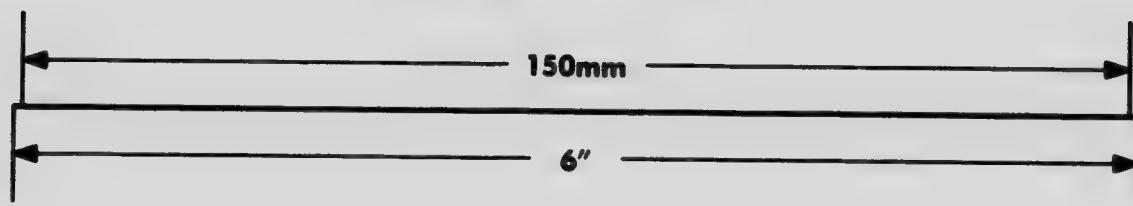
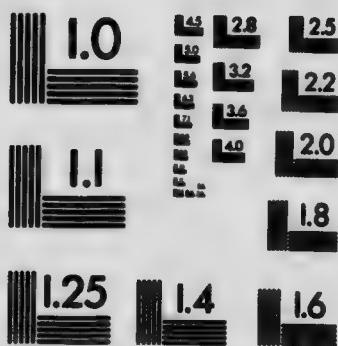
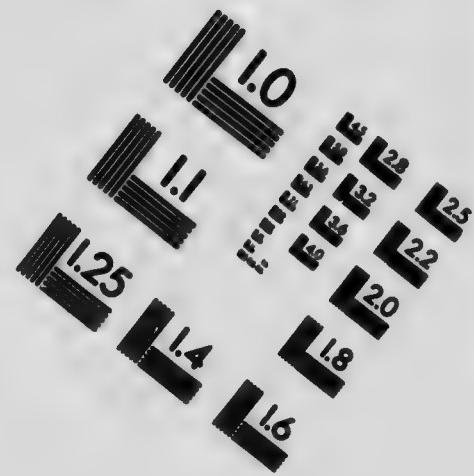
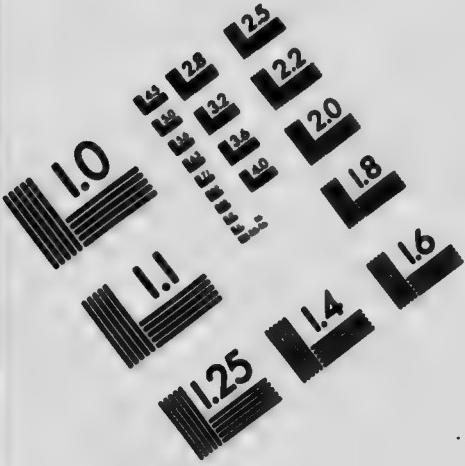
**2. Diseases of the Respiratory Passages.**—Contraction of the nasal cavity, pharynx, larynx and trachea arises from thickening of the mucous membrane, polypous proliferation, changes in the bones, external pressure (struma), the process of cicatrization (after tracheotomy), and also from paralysis of the muscles. The last is the cause of whistling asthma in horses, in which the crico-arytenoideus posticus and consequently the vocal chord, is paralysed on the left side. (See Vol. II., page 690).

**3. Diseases of the Heart.**—The most important are non-compensated valvular defects, dilatation and congenital defects in the ventricular septum. Owing to the disturbance of circulation in the lungs to which they give rise, they produce the so-called "cardiac" pursiness.

**Asthma.**—So-called nervous or intermittent pursiness is generally believed to be a spasmodic difficulty of breathing, which is chronic, but yet has periods almost free from dyspnoea, alternating with occasional spasmodic attacks. This would make this form of tight breathing identical with the chronic asthma of man. More careful investigation into this "nervous" pursiness is much to be desired, especially as the conception of human asthma has not yet been fully cleared up. There are many opinions as to its causes, which shows that the term is only a collective name. The following processes seem to lie at the root of

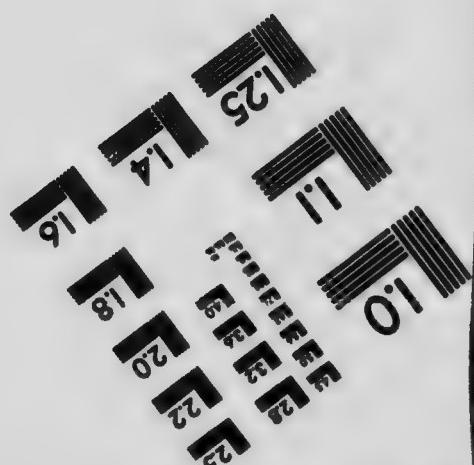
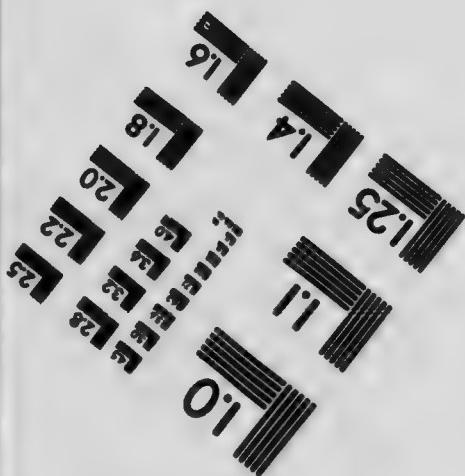


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the malady : 1. Spasm of the bronchial muscles ; 2. Spasm of the diaphragm ; 3. Acute exudative bronchiolitis ; 4. A vaso-dilatating neurosis, in which, owing to nervous causes, great hyperæmia of the bronchioli-walls with stenosis of their lumen is set up. This, which is probably the most important form of asthma, is reflexly caused by a predisposing increase of sensitiveness on the part of the respiratory nerves, and occurs in a great many pathological processes of the nasal and pharyngeal cavities. But there must always be an individual predisposing irritability of the nerves or membranes.

It is very probable that in horses and other domestic animals similar breathing spasms occur, but the question requires to be more fully enquired into. We believe that in dogs we have seen a few cases of genuine asthma, i.e., transient spasmodic dyspnoea, without any demonstrable morbid conditions in the lungs or larynx. Prümmer has noticed the same in a horse, which suffered from a very painful swelling of the right upper maxilla and of the nasal mucous membrane. The dyspnoea abated as the tumour grew less. This very interesting case forms an analogy to the morbid processes of the nasal cavity which so often cause asthma in man, and which can mostly only be surgically removed. Cases have also been observed in cows.

**Symptoms.**—The chief phenomenon in asthma is the difficulty of breathing, which manifests itself in various ways.

1. *Greatly accelerated respiration.* In a broken-winded horse the number of respirations will increase after rapid movement for five minutes to 50-60 and more (instead of about 30), and after movement lasting 30-60 minutes to 80 or 100 (instead of 50). The breathing may also be quite superficial.

2. *Distinctly laboured breathing.* The dyspnoea is thereby much more marked in expiration than in inspiration. The former is characterised by a striking undulating or pumping action of the abdominal muscles, which contract usually in two catches, the first one short and the other longer, producing thus the so-called asthmatic furrow (double lift) behind the false ribs. At the same time there is an active movement, a rise and fall, of the flanks, with projection of the anus. Moreover, expiration lasts disproportionately longer than inspiration. Inspiratory dyspnoea manifests itself in distension of the nostrils and, in whistling asthma, also in the occurrence of inspiratory stridors and much heaving of the ribs. Expiratory dyspnoea represents a loss of elasticity in the lungs, and the inspiratory a contraction of the respiratory lung-surfaces and impediment to the entrance of air.

3. *Slow calming of respiration.* Whereas normally after half an hour of exercise the speed of respiration becomes slowed down again in ten to twenty minutes, or after an hour's

running in twenty to thirty, an asthmatical horse will require respectively half an hour, or an hour and more.

When at rest asthmatical horses often have a dull, faint cough, combined sometimes with ejection of sputum. Percussion and auscultation yield nothing positive. We have, however, in many cases of broken-winded horses detected a distinct diminution of heart-sounds (the heart being covered by the emphysematously enlarged lungs). Along with dyspnoea we may note, especially in the so-called cardiac form arising from heart defects, an intensely laborious action of the heart. Its beat is often palpitating and jerky, more rarely it is imperceptible; the pulse is very much accelerated (80 to 120 a minute), and at the same time irregular and mostly very weak; the heart-tones correspond with the valvular affection and are not clear, but accompanied by after-sounds. Finally, the animals sweat very easily, and these outbreaks of perspiration may be quite extraordinary. General condition is frequently unfavourable, and the hairy coat looks rough and lustreless.

**Course.**—This is chronic and, according to season, weather, food and work done, is marked by improvement and aggravation. The degree of asthma is very varying; it changes from conditions hardly differing from the normal, and in which the serviceability of the animals is scarcely impaired, to such as render them absolutely useless for the time being.

**Therapeutics.**—The remedy most frequently prescribed for asthma is arsenic in small doses continued over a long time, say 1 $\frac{1}{2}$  to 8 grains; or in the form of liquor arsenicalis (1 $\frac{1}{2}$  drams to 1 $\frac{1}{2}$  oz.). An opinion as to the action of this drug is not possible, as accurate observations are lacking. The reports as to its operation vary most strikingly. Some claim that it produces much improvement, others that it is useless, and others again that it is even injurious. That arsenic after long administration can improve an animal's condition and appearance has been fully proved by experience. But that it can also successfully combat emphysema and dyspnoea has yet to be demonstrated. Probably its alleged efficacy has been based upon the improved condition of nourishment. While passing over other remedies which are often commended, we would call attention to a treatment which, according to Garsault (*Parfait Maréchal*), was highly praised as long ago as 1755, viz., the withholding of drink as much as possible. This, as is well

known, plays a great part in the newer treatment introduced by Oertel for chronic disturbances of circulation. The smaller the fluid contents of the body, the more easily the heart works. The withholding of beverages would therefore be the first thing to recommend for so-called cardiac asthma, and in the second place also for all forms of tight breathing which are complicated with disturbed circulation. One might also give the alkalis for chronic bronchial catarrh, especially in the form of artificial Carlsbad salt (3 oz. three times a day, mixed with the fodder). Dietetically one must avoid bulky and dusty foods. Nutrition should be given in concentrated form and all food should be damped.

**The Period of Warranty for Asthma** is in Bavaria, Württemberg, and Baden, 14 days; in Saxony and Austria, 15 days; in Switzerland, 20 days; and in Prussia and Brunswick, 28 days.

## CHAPTER XXV.

### DISEASES OF THE PLEURA.

#### INFLAMMATION OF THE PLEURA. PLEURISY. PLEURITIS.

**Etiology.**—Inflammation of the pleura occurs primarily as an independent disease, but more frequently it appears secondarily in the course of other maladies.

*Primary* pleuritis is met with especially in horses and dogs. It is in many cases caused by cold, so-called rheumatic pleuritis. That cold can produce it has been established by many positive observations. The old report of Duvieusart, who saw 200 sheep stricken with pleuritis after being shorn during a very cold February, best illustrates this statement. But we must admit that in not a few cases of rheumatic pleuritis no previous chill can be proved. Schindelka has frequently observed it in horses after long and uninterrupted transport by rail. Sometimes it almost seems as though pleuritis broke out enzootically under the influence of infectious or atmospheric causes. Youthful animals seem especially liable to attack. Of 600 sucking lambs, Immelmann saw 200 perish of pleuritis. But even strong and vigorous animals, such as good draught-horses, not seldom suffer from pleuritis, perhaps owing to over-exertion combined with chill.

Inflammation of the pleura may occur *secondarily* as a complication of various primary diseases. Inflammation of the lungs produces it most frequently, and particularly the infectious pneumonia occurring during the contagious pleuro-pneumonia of horses and cattle. But genuine croupous pneumonia, especially in cattle, is often associated with pleuritis; the same applies also to catarrhal pneumonia, gangrene of the lungs, and to new growths, particularly cancer, in the lungs. Moreover, new growths of the mediastinum may induce chronic

pleuritis. Sometimes in the course of strangles we observe acute inflammation of the pleura in connection with a mediastinal abscess. New growths, diverticules and injuries to the gullet are very often followed by a pleuritis of ichorous character. In the same way an inflammation of the pericardium may spread to the pleura, especially if there be some foreign body present, as may also inflammation and suppuration in the bones which surround the lungs (sternum, ribs, vertebral column). Indeed, peritonitis may cause pleuritis through the lymph-canals of the diaphragm. Another secondary form is the traumatic pleuritis which arises after contusion of the thorax, penetrating wounds in the breast, fractured ribs, punctures, etc. Finally, pleuritis occurs metastatically in pyæmia and septicæmia, in acute articular rheumatism and chronic nephritis. Tuberculosis and glanders may likewise lead to the development of pleuritis.

**Pathological Aspect.**—From a pathological standpoint we can distinguish a series of pleuritic forms, such as the localised and the diffuse, the dry and the exudative, also adhesive pleuritis; and further, according to the character of the exudation, we have fibrinous, sero-fibrinous, serous, purulent, hæmorrhagic and ichorous pleuritis.

On the outbreak of pleuritis the sub-pleural vessels are much injected, the lymphatic-vessels of the pleura distended, the pleura itself is often infiltrated with hæmorrhages, and is then variously spotted with red, being also lustreless and opaque. A little later the pleura becomes covered with fine, yellowish-white layers or shreds of fibrinous exudate, which can easily be wiped off, they consist mainly of fibrous matter, cast-off endothelial cells, white blood-corpuscles and cell-germs. If the inflammation remain at this stage, there is no further fluid exudation, and we speak of it as dry pleurisy. If no resorption of the dry exudate take place, the exuded layers gradually change, with transformation of their white blood-corpuscles to fibroblasts and connective-tissue cells, into an inflammatory granulation-tissue containing numerous vessels, which change leads to cicatrization and thickening, as well as to adhesion of the two pleural leaves or layers (adhesive pleuritis). In localised pleuritis small white spots (*macula lactea*) and nodules are then formed, as well as tendinous bands and threads between the lungs and ribs; and in the diffuse form, larger membranous formations, with thickening of the pleura. When the inflamma-

tion lasts for long, we may have indurations of exudate over an inch in thickness. These consist of swollen and degenerated tissue and may finally calcify.

Among exudative pleurites the sero-fibrinous form is commonest. In this form a very considerable quantity of fluid may be secreted in a few hours. In horses we find on an average from 4 to 11 gallons in both cavities of the pleura combined. In one case as much as 155 lbs. was present. In dogs the average quantity is from a pint to a gallon, in pigs from  $1\frac{1}{2}$  quarts to  $2\frac{1}{2}$  gallons. The colour of this exudate varies, being yellowish-white, or yellowish-red, or yellowish-green; in the serous fluid flaky or flat fibrous matter is found suspended. On resorption the serum only is at first removed; the fibrinous constituents must first suffer fatty and mucous metamorphosis, or else they are drawn into sacs and remain behind, or calcify. The purely serous form is much more rare.

*Purulent pleuritis* (empyema) is seen most frequently after wounds, infectious conditions of the pleura and in metastases, e.g., during pyæmia. The exuded matter consists here chiefly of white blood-corpuscles. Resorption only occurs in exceptional cases. The empyema rather breaks its way through the lung into a bronchus and discharges outwardly, thus frequently admitting air to the pleural cavity (pyopneumo-thorax); or else the pus works a path through the thoracic wall to the outside (empyema necessitatis). When the pus has been thus discharged, such conditions heal by filling up the pleural space with new growths of granulation and connective tissue.

*Ichorous pleuritis* is developed especially on entrance of air to the pleural sac from the bronchi or thoracic wall, also after the opening of lung-abscesses and lung-caverns in the thoracic cavity. We also frequently meet this form of pleurisy in the course of gangrenous pneumonia, particularly in the contagious pleuro-pneumonia of horses, and it is distinguished by the bad smell and discoloured quality of the exudate, as well as by the presence therein of many kinds of putrefactive-bacteria.

*Hæmorrhagic pleuritis* is seen during hæmorrhagic diathesis or during defective resorption of the fibrinous exudate, when a large formation of new blood-vessels in the fibrin takes place, giving rise to hæmorrhage. The exudate is here of a brown-red colour and rich in red blood-corpuscles (hæmato-thorax).

When there is an abundant accumulation of fluid, the lungs are pressed upwards against the vertebral column, are constricted, diminished in volume and finally become atelectatic

and of tough, hard consistence. The sound parts of the organ are correspondingly hyperæmic or emphysematous. In consequence of one half of the lungs becoming smaller, the respective half of the thorax may also grow smaller and sink in. The heart is thrust from its place, the larger vessels in the thoracic cavity are compressed, so that congestion is set up in the venous system (congested liver, kidneys, etc.), and the diaphragm is pushed back against the abdominal cavity. Pleuritis occurs more frequently on one side than on both.

**Symptoms.**—Rheumatic pleuritis usually begins with sudden shivering fits and great rise of temperature, sometimes also with other rheumatic symptoms, e.g., with colic or evidences of pain in walking. The fever, which at first is somewhat high (up to 105° or 106° Fahr.), becomes later more moderate (103° to 105°), and differs from that of certain pneumonias by its irregular and intermittent character; not unfrequently we observe feverless intervals of several days. The pulse-beat is usually remarkably rapid, which arises probably from pain and also, later, from the pressure exerted by the exudate upon the heart. In horses it fluctuates between 60 and 100 beats a minute, the pulse itself being very weak and small. At the same time there is great lassitude, appetite is poor, external temperature irregularly distributed, the thoracic wall on the affected side being often perceptibly warmer; the tarsal conjunctivæ are very red. In rarer cases the disease develops gradually and slowly, so that on making a subsequent careful examination one is surprised to find an advanced stage of dulness, etc.

Alongside the feverish rise of temperature, the breathing also appears to be characteristically changed. In accordance with the extent of the inflammation it is quickened and impeded, the type of respiration being chiefly abdominal; for whereas the ribs remain almost motionless, the movement of the abdominal walls is greatly increased. Breathing, which is sometimes accompanied by plentiful exudation, amounts occasionally to actual pumping, with a to-and-fro movement of the entire trunk. The nostrils are widely dilated, the fore feet planted far apart, or one in front of the other, the head is often rested upon some support, the animal groans in turning or when the inter-costal spaces are pressed, and either does not lie down at all or only on the affected side. There is also usually a painful cough, but without sputum; this may, however, be

absent. A physical examination of the thoracic cavity is of great importance for identification of the disease. Externally one may sometimes perceive an increased circumference on the affected side of the chest, with a flattening or even a bulging-out of the spaces between the ribs; the heart-beat is frequently displaced. In left-side pleuritis palpation reveals especially a dislocation of the heart, its beat having disappeared from the usual side and can be felt on the right side, especially in smaller animals.

At the outset of the disease *percussion* shows no changes and, even when a fluid exudate has already gathered in the pleural cavity, its presence in the lower third of the cavity can only be proved with difficulty, owing to the muscles of the chest in horses, and on the left side, because of the normal dulness caused by the position of the heart. On the other hand, when exudation is far advanced, the marked dulness of the percussion note in a horizontal direction is characteristic of the pleuritic exudate, as is also the circumstance that the upward horizontal limit of dulness varies with the animal's change of position, even if not in every case very quickly or very completely. The increased resistance also which is noticed in percussion of the thoracic wall is likewise important for the detection of the pleuritic exudate. One sometimes obtains a tympanitic percussion-note at the beginning of pleuritis, when the elasticity of the lungs is partially lessened by a smaller quantity of exuded fluid, and later, often above the horizontal line of dulness, because the lungs, being also pressed upwards by the liquid, have lost something of their elasticity. The dulness rarely rises higher than two-thirds the height of the chest; and the entire pleural cavity is never filled by the exudate.

*Auscultation* reveals, both at the beginning and towards the end of pleuritis, the rubbing sounds (scraping, stroking, friction, creaking and rubbing at intervals), characteristic of the same, and which can often even be felt with the hand. With the advent of the fluid this rubbing noise disappears, and is indeed absent in all cases of a mainly fluid-exudative character. The vesicular breathing sound also vanishes as the pleuritic exudate increases, so that one hears either an indistinct breathing, or nothing at all, or else it is finally replaced by a bronchial breathing when the lungs are only moderately compressed by the liquid; but this also is absent when such compression becomes too strong, because then the bronchi also are constricted. Some-

times one can likewise hear a bronchial breathing above the exudate, i.e., above the horizontal line of dulness, in consequence of compression of the lungs, and this both during inhalation as well as expiration. The vesicular breathing noise in the sound half of the lungs is intensified; and in severe cases the ear may detect symptoms of oedema of the lungs (rustling, rattling). Along with these noises proceeding from the pleura and lungs, one may sometimes notice a dull muscular sound caused by contraction of the inter-costal muscles or of the great pectoral muscles.

Important also are the *changes in the urine*. As the exudate grows in quantity, the amount of urine becomes diminished; it loses also its chlorides. Later, as the exudate is resorbed, the chlorides reappear in the urine, while the latter becomes more plentiful, occasionally very abundant (critical discharge of urine). It moreover often contains albumen; but, according to our observations, the urine of horses often retains its alkaline reaction for a very long time, a fact we must maintain in opposition to the statements of others.

The above symptoms only occur in widely diffused pleuritis. Localised, dry pleuritis occurs almost without symptoms, as is shown by the result of *post-mortem* examination.

**Course and Termination.**—So long as rheumatic pleuritis has not gone too far, its course is usually in so far favourable that complete and rapid resolution may set in. But widespread inflammation of the pleura may, on the contrary, not seldom bring death in its train, owing to oedema of the opposite half of the lungs or to compression of the heart. But in general, pleuritis takes rather a dragging course, broken by sundry relapses, whereby, after a long stage of convalescence, permanent breathing difficulties make their appearance, owing to adhesion of the lung to the pleura as well as to the induration of the lungs which frequently remains behind. Or else the pleuritis becomes chronic, numerous feverish relapses occur, during which chronic disturbances of circulation develop in the heart beneath the pressure of the exudate, as well as in the venous system of the body, with subsequent symptoms of dropsy, which finally pass into general emaciation and cachexia. In extreme cases the exudate at last becomes purulent, and empyema takes place; which announces itself by repeated shivering fits, a high and hectic fever, possibly also by complication with pneumonia (pyopneumo-thorax) and rapid

collapse. Taking all things together, the *prognosis* of pleuritis is not so favourable as that of croupous pneumonia.

**Diagnosis.**—The diagnosis of pleuritis is assured by discovery of the rubbing sounds, the horizontal and distinct dulness, increased resistance under the percussion-hammer, abdominal breathing, diminished or dislocated heart-beat, the absence of nasal discharge and by the atypical and irregular course of the fever. In suspected cases of exudative pleuritis an exploratory puncture will remove all doubt. Although diagnosis is not difficult when the disease is far advanced, yet at its out-break it is often difficult or even impossible to differentiate, especially from pneumonia. In this case repeated and frequent examination by day is necessary, as the rubbing noises are often only heard for a short time at once. For the other signs which distinguish pleuritis from pneumonia, see the latter.

**Therapeutics.**—In contrast to pneumonia, with its typical course, pleuritis always demands medicinal treatment! This may be undertaken in several ways. At the outset cold packing or irrigation of the thorax is best; horses, for example, may be placed underneath a properly arranged irrigator. Strong skin-irritants upon the affected side of the chest have a similar effect, and we recommend for horses either a mustard-plaster or dilute etherial oil of mustard (1:12-20 spirit, oil of turpentine, ether). By these not only is temperature reduced, but the hyperæmia is drawn away outwards, pain assuaged and resorption of the fluid exudate is accelerated. Such inunctions, it is true, generally delay percussion for some days. Where the fever is very intense administer the usual remedies: anti-febrine, anti-pyrine, etc., and especially salicylate of soda and digitalis, the two latter because they also act as diuretics. Should resorption of the exudate be delayed, one may try to expedite it either by dry fodder or by diuretic, drastic, sialagogic or diaphoretic remedies. Besides digitalis (dose for a horse  $\frac{1}{2}$  to  $1\frac{1}{2}$  drams; for a dog  $1\frac{1}{2}$  to 3 grains), the alkalis are also used as diuretics, especially acetate of potash, juniper berries and spirit of turpentine; among drastics calomel was formerly much in use, and has been shown by later observers to have also a diuretic action. Trasböt, for instance, gives horses an internal dose of 30 grains of calomel, and rubs in grey ointment externally. To produce salivation pilocarpine is most suitable (horses  $1\frac{1}{2}$  to 3 grains; cattle 3 to 6 grains; dogs up

to  $\frac{1}{2}$  grain), also arecoline (horses 1.25 grain), subcutaneously applied. A simple Priessnits' compress may also assist resorption of the exudate.

*Surgical treatment by means of puncture is also very important for exudative pleuritis.* Later experience shows that it is well to effect this as soon as possible. The following are the indications for this operation, which, if performed with due aseptic precautions, is quite without danger: 1. When there is danger of immediate suffocation owing to the great quantity of exudate; 2. When the latter generally is very abundant; 3. When the exudate obstinately resists resorption; 4. When it is purulent or ichorous in character. In the latter case a simple puncture does not suffice, but an incision or excision of the rib must be undertaken and the pleural cavity thoroughly drained. Any injury done to the lung during puncture is of small importance. The injection of iodine into the pectoral cavity after puncture, which was formerly usual, has now been generally abandoned.

#### HYDRO-THORAX. PECTORAL DROPSY.

**Etiology.**—This malady, which occurs in all domestic animals, and especially in dogs, is entirely secondary, and is only described here in a separate section in conformity with custom. In contradistinction to "inflammatory pectoral dropsy," as inflammation of the pleura was formerly called, it is also called "chronic" dropsy of the chest. Its causes are congestion of blood after chronic diseases of the heart, lungs and kidneys, especially valvular defects; general dropsy following some anomaly of the blood; and also increased permeability of the vessels. Along with hydro-thorax we therefore often find hydro-pericardium and ascites. Respecting the accumulations of fluid in chronic pleuritis and in sarcomatosis and carcinomatosis of the pleura, as well as in lympho-sarcomatosis, consult the respective chapters.

**Pathological Notes.**—In contrast to pleuritis, hydro-thorax is always bilateral. The fluid collected in the pleural cavities is of clear, serous character, of yellowish, or sometimes reddish colour and without any fibrinous coagulae. The pleura is smooth, and only somewhat loosened by maceration caused by transuded matter. The lungs are compressed in various

degrees, the diaphragm pressed backwards and the inter-costal spaces forwards.

**Symptoms.**—The chief sign of pectoral dropsy is difficulty of breathing; to this must be added physical proof of the transudate. Fever is entirely lacking. As in pleuritis, the animal's breathing is very strained, but costal respiration is not suspended, because the process is neither painful nor inflammatory. Here also percussion reveals a horizontal dulness, which, especially in small animals, at once changes place as they shift their attitude or position. Beneath the line of dulness breathing sounds can often not be heard, but sometimes bronchial respiration above it. Besides these we find also the symptoms of the primary disorder, which is most frequently a valvular defect (after-sounds with the first or second heart-tone; confluence of both cardiac tones into one), or other dropsical conditions.

**Differential Diagnosis.**—Hydro-thorax may be known from pleuritis first of all by the absence of fever and inflammation, of the rubbing sounds, of pain in the thoracic wall and also of cough. Moreover, the flow of liquid in hydro-thorax is always on both sides, while in pleuritis it is often only on one side. This fluid changes its position more easily and quickly in hydro-thorax. Finally, the pleuritic exudate is mostly opaque, contains fibrin and is very rich in albumen, whereas in pectoral dropsy it is clear, without flakes of fibrin and comparatively poor in albumen. But in spite of these differences, it is sometimes hard to distinguish hydro-thorax from chronic pleuritis, especially when a chronic exudative pleuritis gradually changes into pectoral dropsy. In such cases an exploratory puncture is imperative. The probability of an inflammatory process increases whenever the liquid obtained by puncture contains more than 3 per cent. of albumen and exceeds a specific gravity of 1016.

**Therapeutics.**—These are confined to treatment of the primary disease. Along with a dry diet, diuretics are most frequently administered, for dogs especially digitalis or diuretin. Digitalis is either prescribed alone or in combination with acetate of potash and juniper. Diuretin is given to dogs every three hours, 4 to 15 grains in the form of powder, or in combination with digitalis, strophantus, juniper and acetate of

potash. We have also found calomel serviceable with dogs (5 doses per diem of  $\frac{1}{2}$  to  $1\frac{1}{2}$  grains). In hydro-thorax puncture should only be made when life is endangered, as the transuded matter is quickly renewed.

#### PNEUMO-THORAX.

**Etiology.**—By this term we understand the penetration of air to the pleural cavity. If there be also a pleuritic exudate, we speak of hydro-pneumo-thorax, and in case of simultaneous empyema, of pyo-pneumo-thorax. The causes of pneumo-thorax are various. Most commonly air penetrates from the lungs to the cavity of the chest, thus, e.g., by lung-abscesses which break through into the pleural cavity and communicate with a bronchus, or when one of the lung-alveoli is broken, as is observed in cattle after interstitial empyema. In one case of an ox Deupser was able to trace pneumo-thorax to the rupture of an echinococcus-cyst on the surface of the lungs, which communicated with several bronchi. Stray foreign bodies, such as a grain of rye, may also finally pierce the lungs and produce pneumo-thorax. More rarely the air gains access from without through an injury to the wall of the thorax or from the gullet when the same has been torn. On the other hand, pneumo-thorax is not rarely caused in cattle by perforation of the second stomach and diaphragm by sharp foreign objects penetrating to the thoracic cavity. Finally, the generation of gas from purulent exudations in the completely closed pleural cavity may give rise to pneumo-thorax.

**Pathological Aspect.**—True pneumo-thorax is rare, and really only occurs in interstitial lung-emphysema of cattle. The gas here found in the pleural cavity is atmospheric air. More frequently we at the same time find serous or purulent liquid in the thoracic cavity; the air then contains but little oxygen, but much carbonic acid, carburetted hydrogen and sulphuretted hydrogen (gases of putrefaction). The lungs are compressed by this air, the heart is dislocated and the diaphragm pressed backwards.

**Symptoms.**—The signs of pneumo-thorax are a suddenly developed and extreme difficulty of breathing, fall of bodily temperature and symptoms of collapse. The affected half of the thorax appears enlarged, percussion reveals tympanitic

and amphoric sounds, and sometimes also that of the *olla rupta*; if the distension by air be very great, the tone again is loud, or has a metallic ring. On auscultation we sometimes hear as characteristic sounds a metallic splashing, amphoric respiration, as well as a noise of dropping; but in other cases we perceive nothing, or an indistinct and occasionally bronchial breath.

**Therapeutics.**—The air can only be removed by puncture, but the cavity is often refilled with air from the lungs. At the same time the animal's weakness must be symptomatically combatted with stimulants (camphor, hyoscyamine, caffeine, ether, alcohol).

**Forms of Pneumo-thorax.**—In man we distinguish three forms: 1. Open pneumo-thorax, in which the air can stream in and out unhindered through a large opening. 2. Valve pneumo-thorax, in which the air, having entered the pleural cavity, partly closes the opening, so that more air can come in, but can only in part escape again. 3. Closed pneumo-thorax, in which the air, once entered, shuts the opening completely, so that no more can follow.

## CHAPTER XXVI.

### APPENDIX TO DISEASES OF THE RESPIRATORY ORGANS.

#### (A). NON-PARASITIC RESPIRATORY DISEASES OF BIRDS.

Among the various kinds of poultry a simple, primary nasal catarrh is common, which arises from cold and inhalation of irritant matter, and is generally known as "Pip." It is marked by sneezing, snorting, a whistling, rattling breath, opening the beak, slight dyspnoea, shaking of the head, and by a muco-purulent nasal discharge. The presence of this last can be proved by slight pressure upon the nostrils, but very often it dries up within the same to a crust. This unimportant disorder seldom demands treatment. In obstinate cases an addition of alkalis (Carlsbad salt) to the drinking-water may be prescribed. But as a precaution it is always important in cases of nasal catarrh to make sure there is no diphtheria present. In addition to rhinitis, poultry suffer from laryngitis, tracheitis and pneumonia (croupous and catarrhal). Of especial importance is *genuine croupy pneumonia*. *Post-mortem* in such cases, according to Zürn, reveals great congestion of blood in the lungs, hepatization, sub-pleural ecchymosis and extravasation, as well as repletion of the bronchi with thick, fibrous exuded matter. The symptoms consist mainly in laboured breathing with widely gaping bill, signs of pain on touching the thorax, cough and occasional expectoration of an orange-yellow mucus from beak and nostrils. In carrier-pigeons, after traversing long distances, emphysema of the skin occurs, probably in connection with lung-emphysema. But it usually passes off after a few days' rest (André).

#### (B). PARASITIC RESPIRATORY DISEASES OF BIRDS.

##### I.—POULTRY-DISEASE CAUSED BY SYNGAMUS TRACHEALIS.

**Natural History.**—The poultry-disease described below was first outlined by Wiesenthal in 1797 in America, where it wrought great havoc. It appeared in England in 1806, where ever since, and also in France, it has been one of the most destructive diseases of birds, especially in preserves of pheasants

and poultry-yards. Year by year more than a million fowls perish of this disease in England alone. In Rothschild's pheasant-preserve in France it is reported by Mégnin that, during the prevalence of the plague, 1,200 birds were found dead every morning.

The paired wind-pipe worm (*syngamus trachealis*) belongs to the class of strongylides and occurs in the pheasant, turkey, domestic fowl, partridge, peacock, gosling and also in many other kinds of bird, such as the stork, popinjay, starling, weaver-bird, magpie, crow, swift and jackdaw. It lives in sexual maturity in pairs, the male copulatively joined to the female, in the upper part of the trachea, and mostly just beneath the rima glottidis. The female is  $\frac{1}{2}$  inch long, and the male  $\frac{1}{3}$  inch; their colour red, shape cylindrical, and diameter about  $\frac{1}{5}$  inch. Both members of the pair attach themselves by a cup-shaped suction-dics to the mucous membrane, whence they suck blood and thus set up tracheitis. Larger animals can bear the parasite more easily, but in smaller ones with a narrow wind-pipe, the entire lumen is often so filled up by it that they must choke. The development of the parasite is as follows: The female lays smooth, elliptical eggs, which, on being coughed out, reach the digestive canal and pass out with the dung. In warm weather eel-like embryos are developed in about eight days, the process in winter lasting some weeks. These are taken up by other birds with their food, and reach the trachea in some way at present not exactly known. There they may begin to cause cough after the seventh day.

Walker found *syngamus* embryos in the earth-worms of the district where the plague raged, and concluded that these form the intermediate hosts. According to Salmon, it has not been proved that earth-worms are necessary as intermediate hosts for these parasites; but he maintains that worms merely contain these embryos because they take in earth which happens to hold them. But to Walker belongs the credit of having discovered that earth-worms from a district where the disease is rife can communicate it to fowls which eat them. He recommends destruction of the worms in the soil of a hen-run by strong salt-water (?).

**Symptoms.**—At first the birds appear less lively, cease taking food, often open their beaks as though yawning ("gape"). Then they begin to cough, toss their heads about, by which act whitish lumps are often thrown out. As the cough increases, breathing becomes more difficult, the creatures actually snap for air and either gradually suffocate with con-

stantly growing tightness of breath, or else suddenly and owing to stoppage of the rima glottidis. For diagnosis it is important to prove the presence of the elliptical eggs in the faeces. Recovery only occurs in larger and older birds, or when merely solitary worms have gained entry.

**Therapeutics.**—Prophylaxis consists in prompt separation of sound and diseased birds, as the former may be directly infected by picking up the expectorated masses of worms; also in thorough disinfection of sheds, roosts, etc., and in burning all corpses. Change of diet is also to be commended, with careful cleansing of all feeding vessels. Against the disease itself Mégnin advises a mixture of chopped garlic with the food, and a decoction of the same as drink. Others use inhalations of tar. Mouquet and Cordier recommend the intratracheal injection of a 5 per cent. solution of salicylate of soda as a very good remedy. Sometimes it is possible to remove the worms mechanically by means of forceps inserted through the opening of the larynx. For this purpose Cobbold performed tracheotomy.

**Other Worms of the Wind-pipe.**—Of other parasites which settle in the respiratory organs we must mention monostomum flavum, which occurs in the trachea of ducks; also the thread-worm of pigeons, filaria clava, found in the connective tissue between the anular cartilages of the trachea.

## 2.—CYSTOLEICHUS SARCOPTOIDES, THE AIR-SAC MITE OF FOWLS.

**Natural History.**—This parasite inhabits especially the wind-pipe, bronchi and, above all, the so-called air-cells, especially the abdominal cells of hens and pheasants. Viewed by the unaided eye, the mites are round, white dots, about  $\frac{1}{16}$  inch in diameter. On enlargement we see a sarcoptes-like mite, with tortoise-shaped, oval body, small oval head, and four pairs of legs, each of five members and provided with pedunculated adhesion-discs. There is possibly some genetic connection between this cystoleichus sarcoptoides and the sarcoptes cisticola (see Vol. II., page 508), which occurs encysted in various organs of the abdominal cavity and lives under the skin. The two mites are often found together.

**Post-mortem Lesions.**—The air-sac mite causes violent croupous inflammation in the bronchi, so that these are some-

times filled with a firm, yellowish plug. In the finer bronchi we discover the mite embedded in glairy or slimy mucus, which is often traversed by dots of blood. The lung is in places hepatized and oedematous. Occasionally the mites are found in the abdominal cells without causing any inflammation, the same being literally sprinkled white with them.

**Symptoms.**—The phenomena produced by the presence of air-sac mites consist in accelerated and impeded breathing. According to Zürn, the birds also emit a peculiar tone, as though there were some foreign body in the upper larynx. Otherwise they appear lively and eat well.

**Treatment.**—This is not likely to be of much effect. One must attempt to bring mite-destroying agencies to act upon the parasites through the air inhaled. To this end inhalations of the vapours of tar or tar-water are recommended.

**Cytoditis nudus.**—This mite has been found by Edgar in the bodies of fowls which had died suddenly. The abdominal cavity was covered with a greyish yellow dust, consisting of thousands of mites. They were also present in the heart, pericardium and aorta.

### 3.—PNEUMO-MYCOSIS IN POULTRY.

The invasion by fungi of the lungs of birds (hens, pigeons, geese, ducks, parrots, swans, pheasants and cage-birds) arises usually from the settlement therein of *aspergillus glaucus*, *migrescens* and *fumigatus*, also of *mucor racemosus*. These fungi attain the bodies of their victims by inhalation of dust, or by consumption of mouldy food (millet, etc.). Besides the lungs, they are found in the bronchi, air-cells and nasal cavity. Probably owing to the generation of an inflammatory toxin (phlogosin), they produce upon the mucous membranes a diphtheritic layer in the form of sheets and scales of lamellar structure, as well as thickened, lumpy and caseous masses of pus, which contain the mycelium of the fungus and the conidia. Mycosis is set up in the lungs in nodular form, as in the mammalia.

The essential symptoms of this disease consist in accelerated, snoring respiration with rattling sounds, in diminished appetite, great lassitude (drooping the wings, closing the eyes), in wasting almost to a skeleton and finally in profuse diarrhoea, death

usually following after a sickness of several weeks. Observations made in Belgium and France show that the disease may be transmitted from pigeons to man (breeders of the same), causing a malady like tuberculosis of the lungs.

**Infusoria.**—Willach reports upon an enzootic among pigeons caused by infusoria. The lung-tissue is hepatized and necrotic, the liver and muscles contain yellow nodules, in which are oval, greenish bodies possessing the shape and aspect of the red blood-corpuses of birds. These Willach regards as pathogenous infusoria, and has given them the name of *balantidium viride*.

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